UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF NORTH CAROLINA
ASHEVILLE DIVISION

STATE OF NORTH CAROLINA ex rel. Roy Cooper, Attorney General,	)	
Plaintiff,	)	No. 1:06-CV-20
vs.	)	VOLUME 5A (Pages 1019-1169)
TENNESSEE VALLEY AUTHORITY,	)	
Defendant.	)	

TRANSCRIPT OF TRIAL PROCEEDINGS
BEFORE THE HONORABLE LACY H. THORNBURG
UNITED STATES DISTRICT COURT JUDGE
JULY 18th, 2008

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I N D E X

I N D E X O F E X H I B I T S

FRIDAY MORNING, JULY 18, 2008

THE COURT: All right. Let's have our witness come back around.

MR. GOODSTEIN: All right. Dr. Levy.

JONATHAN LEVY

DIRECT EXAMINATION (Cont'd.)

BY MR. GOODSTEIN:

Q. Good morning, Dr. Levy.

A. Good morning.

Q. Have you come to a conclusion regarding the benefits to public health that will accrue if TVA reduces its air pollution emissions as North Carolina is seeking in this case?

A. Yes, I have.

Q. And can you give us a summary of that conclusion.

A. Sure.

MR. LANCASTER: Note my objection on the scientific reliability grounds.

THE COURT: All right. Thank you.

All right. You may proceed, Dr. Levy.

A. Thank you. So within the health impact assessment that we conducted, this involved combining the emissions estimates from Dr. Staudt and the atmospheric modeling from Messrs. Chinkin and Wheeler. Once we combined that with information on the health effects of fine particulate matter on ozone, population data and health outcome data, health

incidents data, we were able to estimate public health impacts in the states surrounding the power plants and across the region.

From our experience in conducting previous health impact assessments of power plants, I would conclude that the impact of what have been termed the excess emissions from the TVA plants are substantial in magnitude and are contributing and would contribute to both mortality effect and morbidity effect or nonfetal illnesses, again, throughout the region in which the atmospheric modeling was conducted.

Q. And you also reached a conclusion regarding current impact to public health resulting from excess emissions from TVA's coal-fired power plants?

A. Yes, I did. The conclusions regarding the current impacts are very similar to the benefits if the emissions were reduced. It would include the benefits as well as the impact from the residual emissions, so the current impacts would be certainly larger than the values that we calculated. But the methodology is identical and the qualitative conclusions are identical.

Q. Dr. Levy, can you identify the reports that you and Dr. Spengler prepared in this case. They should be in the back of your notebook, Plaintiff's Exhibits 469, 470 and 471.

A. Yes, I can. These were the reports we prepared.

MR. GOODSTEIN: Your Honor, we offer 469, 470 and

471 into evidence at this time.

MR. LANCASTER: Your Honor, that's subject to the objection on the scientific reliability grounds. And it's further subject to the objections, sir, that these reports purport to quantify health impacts such as mortality in a number of states that I understand Your Honor's ruling ruled are not in issue, states such as Wisconsin and New York, far away states.

Those are our two objections to the reports. We certainly don't mind the court having access to the reports.

THE COURT: Okay. Show the objections and the court ruling of overruled.

All right. Proceed.

BY MR. GOODSTEIN:

Q. Dr. Levy, in order for us to understand your analysis, it would be helpful if you could give us an overview of the methodology that you used, the health impact assessment methodology.

A. Sure. So health impact assessment as we have done it is really a special case of risk assessment as a whole which I described a little bit yesterday, but as a reminder has been codified as a four-step process involving identification of hazards, exposure assessment, dose response assessment and then risk characterization. These are the four steps classically defined by the National Research Council in their

1983 Red Book report. And then the work that I conducted could be considered as an application of these risk assessment methods.

If it would be helpful, I would often find this helpful when I give one of the lectures in my risk assessment class, I can draw the diagram that shows the steps in the risk assessment process and how that ties into the efforts that we conducted.

MR. GOODSTEIN: With your permission, Your Honor, if we could have Dr. Levy approach the pad, I think that would assist us in understanding his methodology.

THE COURT: All right, you may do so.

(Witness stepped down from the witness stand.)

THE WITNESS: I will apologize in advance that my handwriting is poor and this pad is somewhat small so I may need to abbreviate a few things, but if you'll indulge me.

As I mentioned, risk assessment involves four steps and I'm going to portray that as four boxes with connected arrows here.

So the first step, as I mentioned, is called hazard identification. So I'm going to write that as shorthand as hazard ID here. And what that is defined as is a qualitative step to basically consider do the pollutants that are being considered in the risk assessment appear to have health effects in the human population at the levels of exposure that

are being considered? So it's a qualitative step to determine if the quantitative steps will be necessary.

The second step is exposure assessment. And this is generally defined as either measurement or modeling of exposures to human populations. In a case like this, as I'll talk about in a little bit of detail in a moment, it involves modeling because clearly we can't measure the impact in 2013 so we need to model what those impacts would be.

The third step is what is known as dose response assessment, or in this case for air pollutants where we're taking modeled estimates for the ambient air. It's also called concentration response assessment. So at times I'll use concentration response functions or dose response functions which are not exactly interchangeable but in this context are similar.

And the last step involves what is called risk characterization, which I can't fit in the box, but that is meant to say risk characterization. And that basically involves combining the exposure assessment data, the dose response data and population data to arrive at quantitative estimates of health risks.

So as mentioned, this -- this is a paradigm that was really codified in 1983 in the Red Book. It certainly existed before that time. The National Academy Committee came together in '83 to really synthesize the risk assessment work

that had been happening in the 1970s related to asbestos, nuclear power and other applications. But, you know, this was codified in that report and really has been accepted worldwide as the standard approach for risk assessment.

So now on the right-hand side what I'm going to portray is health impact assessment and specifically what we did in this case, so this will be a set of four parallel boxes.

So in the hazard identification step as, you know, I can talk about in more detail, we look through the literature, the epdemiologic literature, the toxicologic literature, the full set of literature on the different potential air pollutants. When we did that, there's certainly a number of air pollutants that could have been considered in this assessment and that are associated with human health, but we focused in on fine particulate matter or $PM_{2.5}$ and ozone. And we arrived at that decision because there's by far the largest literature available on health effects of those pollutants. There's strong and systematic evidence of both the biological plausibility of their effects and evidence available that would let us construct concentration response functions for those pollutants.

So we certainly didn't mean to imply within our assessment that other pollutants such as mercury or sulfur dioxide or nitrogen oxides would have zero impacts, but that

these we felt were the most important to consider.

The exposure assessment step, as already has been alluded to involved looking at what I'll put in shorthand here as delta emissions, which is basically the difference in emissions between current emissions -- or rather, I should say base case emissions for 2013 and the emissions that could be achieved through application of controls as discussed by Dr. Staudt. So that was the first piece in the exposure assessment.

And then that feeds into the CMAQ modeling that was described by Mr. Wheeler and Mr. Chinkin to estimate the changes in concentrations across the model region. And so that represented the exposure assessment piece that was conducted by others in this application.

The dose response assessment involves, as I think I alluded to yesterday, figuring out these concentration response functions or the relationship between changes in concentrations and changes in different health outcomes. We looked throughout the literature for a variety of health outcomes and focused in on a subset of outcomes where the literature was strong and robust. And so this for PM and ozone involves mortality and multiple morbidity outcomes, which are described in more detail in our report.

And then the final step of risk characterization, as mentioned, involves basically multiplying this change in

concentration by the concentration response functions and combining that also with population data, the number of people who live in these different geographic areas and the rates of disease or the rates of mortality in those locations. So unfortunately, this is a small box so I'll have to use more shorthand.

And then the last thing I put here in very much shorthand, U and V, and that says uncertainty and variability. And part of the risk characterization step involves certainly summarizing the risk, talking about the -- you know, obviously calculating the risk including the population data and the incidents data and considering uncertainty and variability. Variability meaning differences across the population, differences in different geographic areas. And uncertainty corresponding to sort of common parlance of uncertainty, things that could lead one to different calculations and different risk estimates.

I think it's important to recognize here up front, I think, two things. One is that risk assessment was developed as an applied discipline and as a discipline to help decision makers make decisions under uncertainty. That is the rationale for the tool and to synthesize evidence to help those decisions be arrived at.

The second thing to recognize is that uncertainty analysis can take many forms. The World Health Organization a

couple years ago laid out that -- they said there are four levels of uncertainty analysis. People can say that there are more or fewer. But essentially, that uncertainty analysis can include qualitative descriptions of uncertainty, what they call sort of range finding calculations, basically figuring out how big or how small could the risk be, all the way to very fancy statistical models of uncertainty. And the key thing that they pointed out and others have pointed out is that there's no one right way to do uncertainty analysis. Uncertainty needs to be considered and addressed, but that the decision context dictates how uncertainty would be considered within the assessment.

So I'll talk through a lot of these things in more detail, but I think this lays out kind of the chain of calculations that we conducted.

(Witness resumed the witness stand.)

MR. GOODSTEIN: Your Honor, that's been marked as Plaintiff's Exhibit 485 for identification and we offer it into evidence at this time.

THE COURT: 485?

MR. GOODSTEIN: 485.

THE COURT: 485.

MR. GOODSTEIN: Yes.

THE COURT: All right. Let it be admitted.

(Plaintiff's Exhibit Number 485 was received into

evidence.)

Q. Dr. Levy, why did you choose to use this type of health impact assessment for this particular study?

A. This is the standard methodology used when you want to understand and to quantify the health benefits of emission controls or the health implications of a delta emissions or a, you know, excess emissions, a change in emissions. This is the approach that EPA uses within its regulatory impact analyses for air pollution regulations that have potentially large economic effects. So they have used this since the mid '90s to look at the benefits and costs of the Clean Air Act, the benefits and costs of the Clean Air Interstate Rule, Off-Road Diesel Rule. It's been applied in the context of the National Ambient Air Quality Standards. So it's been used quite a bit by EPA. It's been used by the European Union, by the World Health Organization, used in global burden of disease assessments, and obviously within the academic literature as well. So it's a very standardized approach and, you know, as hopefully the diagram illustrated, you know, it really is an application of the classic risk assessment approach, you know, focusing in on the questions that are pertinent in this case.

Q. So this method is recognized by many experts in the field of environmental risk analysis.

A. Yes. I mean, the four steps as they're known in

shorthand are, you know, universally accepted within the risk assessment community and then this health impact assessment, which is a special case of those four steps, are also very widely accepted.

Q. Many experts have used this methodology, the health impact assessment methodology?

A. Yes. As mentioned, certainly the USEPA, regulatory bodies in Europe and the WHO. The California Air Resources Board conducts a number of health impact assessments for California specific regulations. The EPA has even developed a software package called BenMAP, B-e-n-m-a-p, that, you know, takes the health impact assessment methodology and puts it in a software package on the web site that's, you know, reasonably user friendly, anyone can download, and then conduct health impact assessments. And that tool by EPA has been used, obviously, by EPA and by a number of state agencies as well.

Q. And the health impact assessment methodology was used by EPA at least as far back as 1997?

A. That's correct. I think -- it was certainly used even prior to that time, but I think in large scale application it started being used, I think, in that 1997 application, in part stemming from President Clinton's executive order requiring, you know, quantitative benefit cost analyses, you know, for regulations that may have large economic impacts.

Q. And can you give us some examples of recent applications of the health impact assessment methodology by USEPA.
A. Perhaps the most pertinent one to this case would be their 2005 health impact assessment of the Clean Air Interstate Rule or CAIR in which they conducted a health impact assessment. Looked at the reduction in emissions that would occur with application of CAIR to power plants in the eastern United States. Used the CMAQ model to quantify the changes in concentrations that would occur. Applied concentration response functions for particulate matter and ozone and then calculated the risk throughout the region -- or the health benefits, I should say, throughout the region to compare with the costs of control. It followed a very similar paradigm to what we conducted within our reports.
Q. Let's talk about your experience with performing health impact assessments. Have you performed them before the one that you did for this case?
A. Yes. On a number of occasions, you know, spanning back about ten years or so within the peer-reviewed literature as described in my CV, the number of publications, developing components of health impact assessment as well as full-bore health impact assessments and then, you know, preparing this report as well as a health impact assessment for a power plant in Wisconsin as part of an administrative hearing.
Q. So based on your experience, this methodology, as you

performed it in this case, is reliable.
A. Yes. It's a very reliable, very well accepted methodology.
Q. In summary, how did you determine that the health impact assessment you performed in this case yields reliable results?
A. Well, certainly following the classic four-step approach ensures that we're not leaving anything out and that we're doing the calculations in a reasonable way. You know, we observed the quality assurance steps taken upstream, taken in the emissions and concentration modeling and, you know, mapped the concentration outputs ourselves to verify the output, so we were comfortable with the input data to our assessment.

You know, we carefully and rigorously read the epidemiologic and toxicologic literature to develop concentration response functions for a variety of health outcomes and in each case arrived at what we considered to be the best estimate to use within a health impact assessment.

We used publicly available databases relied upon by USEPA and many others to determine population data that came from the U.S. Census to determine baseline health outcome data which came from public CDC databases.

And then when we combined all that information together, we conducted standard quality assurance measures to ensure that there were no errors in the calculations as well as comparing the outputs from our assessment with the outputs of

other assessments to ensure that they were reasonable in comparison with what other people had found for corresponding changes in emissions.

So, you know, we, in each case, tried to rely on and did rely on the best available evidence, the strongest databases and the information that others, particularly USEPA, but many others had also utilized in the similar calculations.

Q. So let's look at step one of your analysis, Dr. Levy. You mentioned that you determined that $PM_{2.5}$ and ozone were the pollutants that you want to focus in on in your health impact assessment. Are there significant health effects associated to exposure to fine particulate matter?

A. Yes, there are. And this is something that, you know, there are volumes of published studies in the peer-reviewed literature. Just as an example, the USEPA criteria document for $PM_{2.5}$ which was disseminated roughly three years ago, you know, it was a voluminous 2000-page document with many hundreds of references of many more studies that have come on since that point. And it's, you know, especially across the last ten years, but in reality for probably 30 or 40 years, it's been a heavily studied pollutant with a large amount of literature, and particularly in recent years literature developing the biological mechanisms for health effects and establishing health effects at current levels of exposure.

And so, you know, looking at that large body of

literature which many other expert groups have also looked at and arrived at similar conclusions, we concluded that at current levels of exposure and the levels of exposure projected in 2013, health effects would be anticipated from $PM_{2.5}$.

Q. And you looked at whether these health effects are biologically plausible and connectable to $PM_{2.5}$?

A. That's correct. In the hazard identification step, especially when one is relying on epidemiologic evidence as one does to develop concentration response functions, usually go through a version of what are called the Hill's Causal Criteria, which are a set of criteria laid out to try to determine whether an exposure is causally contributing to a health effect. And, you know, there's a number of those criteria. You know, one is biological plausibility. Others are consistency across the literature, coherence with other known facts about the pollutant, and outcome, you know, the existence of dose response gradients. Basically, you know, when you're exposed to more of something, you see more of an effect and so forth.

And so we went through those criteria and concluded that for fine particulate matter it easily surpassed the hurdle that would be laid out by Hill's Causal Criteria, including biological plausibility, but also including the large consistency of findings across many epidemiologic studies.

Q. And for $PM_{2.5}$, did you confirm the association with premature mortality?

A. Yes, I did. We, in the hazard identification step, looked broadly at the potential for health implications for $PM_{2.5}$; but then, obviously, once it passed that screening test, we need to figure out which outcomes were causally associated with $PM_{2.5}$. And so one of the outcomes that has been consistently and strongly associated is premature mortality in both cohort studies and in time series studies.

And just as a parenthetical, cohort studies are where individuals are recruited into the study and then followed over time and, in this case, to see who dies and to determine whether those deaths are associated with air pollution or other risk factors.

Time series studies look at day-to-day changes in air pollution and day-to-day changes in outcomes like mortality or hospital admissions.

And so there's a very large literature both from cohort studies and time series studies that confirms the relationship between $PM_{2.5}$ and mortality.

Q. And you also confirmed that association between $PM_{2.5}$ pollution and cardiovascular problems and respiratory problems.

A. That's correct. As Dr. Peden alluded to, there's good biological plausibility for both respiratory and

cardiovascular effects of $PM_{2.5}$ and those can certainly run the gamut from a severe effect that would result in a hospital admission down to an asthma attack or a respiratory symptom. And so those effects were considered biologically plausible and we looked at an array of nonfatal, both cardiovascular and respiratory health effects.

Q. And have these relationships been confirmed by the National Research Council and the EPA?

A. Yes. The EPA has, within their regulatory impact analyses, developed concentration response functions for $PM_{2.5}$ mortality relying on the cohort literature as we have done and for a large number of morbidity outcomes, a larger list than we considered in this case. We considered a smaller number of health outcomes than EPA normally considers.

Q. And has the American Heart Association confirmed these relationships of $PM_{2.5}$ pollution and health effects?

A. Yes. They had an article, I believe it was in the journal Circulation, I may be mistaken about that, where a large number of people associated with the American Heart Association essentially said there's strong -- a strong biological basis and biological plausibility for cardiovascular effects of $PM_{2.5}$ and, you know, essentially endorse the need for, you know, further control of fine particulate matter exposures in the United States.

Q. And based on your experience and review, do panel studies

also support the relationship between $PM_{2.5}$ for pollution and these health effects?

A. Yes, they do, and there's very large literature out there, only a subset of which we use to develop quantitative concentration response functions. But the panel studies help us to understand mechanisms and understand biological plausibility and effects. And so there's many other studies like panel studies, like exposure assessment studies that help us understand what could be going on within the epidemiologic literature. Those panel studies are not quantitatively represented within our assessment but they certainly provide corroborative evidence.

Q. And you also mentioned Dr. Peden's testimony and reports in this case.

A. Yes.

Q. Do they support your conclusion about the association between these health endpoints and $PM_{2.5}$ pollution?

A. Yeah, they're very supportive. I think his reports described nicely the mechanisms by which both respiratory effects could occur as well as cardiovascular effects and contributing to disregulation of the heart and so forth which can lead to a cascade, to hospital admissions and premature death. So it's very consistent with our approach and with the epidemiologic literature we incorporated.

Q. Let's talk about those. Can you tell us what significant

health effects that you associated in your work here with exposure to ground level ozone.

A. Ozone has also had a voluminous amount of publications, again a multi-thousand page criteria document from EPA laying out that evidence. And this includes evidence of both premature mortality from time series studies related to short-term changes in exposure as well as morbidity effects, largely focused on respiratory effects, respiratory hospital admissions, exacerbations of asthma or other sorts of respiratory symptoms, and what has been termed minor restricted activity days which is an unfortunate bit of jargon but basically refers to days in which people can conduct some of their regular activities but not all of them. So maybe they can go to work, but they can't go for a run or they can go half a day for work and can't, you know, last the entire day.

Q. And what are the main health effects associated with ground level ozone?

A. Premature mortality has been, you know, consistently associated with ground level ozone. This was really well established in the three meta-analyses that were conducted back in 2005, one of which I was involved in, that all confirmed that the epidemiologic literature was showing a relationship between ozone and mortality.

The National Research Council in their report this year

endorsed that approach and said that there was a plausible causative association between ozone and mortality.

And then ozone is associated with a cascade of nonfatal health effects influencing the respiratory system.

Q. And we heard about that yesterday from Dr. Russell.

A. Yeah, that's correct.

Q. And did you confirm the biological connection and plausibility of these health endpoints with ozone pollution?

A. Yes, I did. Ozone has been a very well studied pollutant for decades and there's good understanding of its effects on the lungs and the ways in which it can contribute to respiratory deficits. And so it's -- there's a large compendium of both panel studies, chamber studies, animal studies, all of which confirm the health effects of ozone, and then epidemiologic studies that reinforce those effects and demonstrate effects at the current levels and projected 2013 levels of exposure.

Q. And does Dr. Peden's testimony in this case and his reports in this case also support those relationships of those health effects that he mentioned and ozone pollution?

A. Yes, they're very supportive of the type of health effects and the nature of effects that we quantified.

Q. Does the recent National Academy's ozone study that came out this year also support those relationships?

A. It mostly focused on the mortality effects and supported

the quantitative approach that we applied and supported that this relationship between ozone and mortality can be put into concentration response functions and used in regulatory assessments.

They also provided some description, if I recall correctly, of the mechanisms by which this could occur, the plausibility of the effects and the likelihood that morbidity effects would be exhibited not just mortality effects. So it is very confirmatory of the approach we used.

Q. All right. Let's talk about step two of your analysis, Dr. Levy, if you will, please, the exposure evaluation. How did you determine the change in exposure to $PM_{2.5}$ and ozone?

A. Well, that -- that, as mentioned, really relied on the outputs from Messrs. Chinkin and Wheeler. They applied the CMAQ model which, you know, certainly is a state-of-the-art model for conducting these sorts of assessments because it can capture the effects of the changes in emissions over a large geographic area, which is the common approach within these health impact assessments.

And so, you know, we relied on the outcomes provided by Messrs. Chinkin and Wheeler which gave these delta concentrations or the effect of the excess emissions within each of the model grid cells from their CMAQ modeling.

Q. All right. I'd like to show you Plaintiff's Exhibit 225 for identification which should be a figure out of your

report, Dr. Levy. It's on the screen as well as in your book behind the next tab.

A. Yes.

Q. Plaintiff's Exhibit 225.

Is this the modeling domain that you considered in your public health impact assessment for this case?

A. Yes, it is.

Q. And can you explain to us why you used this modeling domain.

A. I guess there's probably two responses to that. You know, one is the CMAQ model certainly is able to provide effects of changes in concentrations over a large geographic area.

And the second reason is that our previous studies that we conducted on power plants in Massachusetts, Illinois, the D.C. area and Georgia, in each case we confirmed that quantifying the total health impacts requires a fairly large spatial domain. It can take time for secondary sulfates and nitrates to form in the atmosphere, and when they form they can travel a fairly large distance.

So in some of our earlier papers, we modeled, for example, within a few hundred miles of the power plants and we subsequently demonstrated that that would leave out, perhaps, half of the health impacts in some cases and in some cases much more.

So we certainly concluded that a larger spatial scale would be necessary for a comprehensive health impact assessment and this larger spatial scale made available by a model like CMAQ was therefore the appropriate approach to use.

Q. And this is the modeling domain that CMAQ provided results for.

A. Yes, it is the identical domain.

Q. And have you reviewed the changes in concentrations of $PM_{2.5}$ and ozone that were provided to you by Messrs. Chinkin and Wheeler?

A. Yes. Prior to conducting our assessment, we reviewed their outputs. We developed our own maps of the outputs to reassure ourselves of the reasonableness of the outputs, as well as the spatial patterns of those outputs. And so we confirmed that they, you know, appeared correct, appeared reasonable and were useful for our application.

Q. Let's move to step three of your analysis, Dr. Levy. Can you describe to us how you derived the concentration response functions that you used for your analysis in this case.

A. So for each health outcome, we really approached it fresh. Looked at the totality of the literature available at the time that we developed our concentration response functions and then quantitatively synthesized that evidence.

For ozone, as mentioned in our report, I had recently conducted a health impact assessment in which we developed

those functions just a few months prior to developing the report. So we felt confident that we had the most recent literature available and we had a peer-reviewed and published set of concentration response functions.

So we used those directly and we rederived the concentration response functions for fine particulate matter, including mortality and morbidity, again, by pulling the full set of studies, synthesizing them, evaluating which were applicable within this context, and then developing the quantitative relationships.

Q. All right. Let's use as an example the $PM_{2.5}$ mortality concentration response function.

A. Okay.

Q. Do you have a figure in your report that you can show us which helps you explain how you determined which studies to rely on to develop the concentration response function for $PM_{2.5}$ mortality?

A. Yes.

Q. Let me show you Plaintiff's Exhibit 226 for identification. And can you explain, Dr. Levy, why you included this in your report and what it shows.

A. Well, there's a lot of detail in this table and I'm not going to talk on all the detail, but, you know, big picture.

First of all, we synthesized the literature not from this table but independently. This table is from a paper by Pope

and Dockery in 2006 and provides a nice summary of most of the key cohort studies looking at $PM_{2.5}$ mortality and provides us a way, I think, to demonstrate how we look at the totality of the literature and then figure out from that literature what the best concentration response function would be.

And I'm going to attempt to draw some lines through this on my touch screen to demonstrate the approach we followed. Apologize if it's a little messy because I'm going to have to try to draw small lines.

But you can see there's a large number of publications. You know, in this case I'm focusing on all cause mortality. Those arrows missed slightly, but focusing on the all cause mortality columns, so for the time being we can neglect the two other columns and just focus in on all cause mortality.

The left-hand column shows what the studies were and obviously the next column the reference to the peer review publication. And part of the synthesis process involves not just taking every study available, throwing it into a big soup and coming out with a number. We carefully look at them for applicability within this context. And so part of applicability is the -- having the same kind of exposure measure that we are using in our assessment and the same type of at risk population as in our assessment.

So if we start from the bottom, from the last row, you can see there's a study that is labeled cystic fibrosis. And

that is a study of the effects of $PM_{2.5}$ in patients who have cystic fibrosis. And that is a very valuable study, but is not a generalizable concentration response function to the U.S. population or to the population in our domain.

Similarly, you can see the four studies just above cystic fibrosis starting with Netherlands. Again, I missed a little bit on the line. Thanks for helping out. And all four of those studies, again, valuable studies, but their exposure increments, you can see, are either being near a major road or you can see it says BS which stands for black smoke in this context and that's a measure essentially of diesel-related pollution. That is not directly applicable for our concentration response function. So again, valuable literature. Important in some settings, not in our setting in this case.

Now, if we keep working our way up the list, there's a set of additional cohort studies. For the moment I'm going to bracket these two studies here that are labeled postneonatal infant mortality. Those studies we did use, but for now I'm talking about mortality in adults so we're not going to really address those at the moment.

And then we have the studies that are in between the blue box and the yellow box and each of those studies had an issue that made it not applicable in this case. You know, for example, we have the bottom study by Enstrom which solely

focuses on the elderly population of California and had some -- we had some methodological concerns with that study.

The study above it labeled VA -- or rather, the two studies above it labeled VA are studies of male military veterans with mild to moderate hypertension receiving treatment for that hypertension at VA hospitals. So it's a very specific cohort that is not generalized to the U.S. as a whole.

The two lines above that did not provide concentration response functions for $PM_{2.5}$ mortality and so those were excluded.

And then the final set of studies, AHSMOG studies that you can see here -- for some reason everything is coming out on my screen a little bit higher than where I press it so indulge my slightly errant arrows. This is a study of Seventh Day Adventists in California which is a fairly unique population that generally abstains from smoking and alcohol consumption and so forth, and so that study is also not generalized to the U.S. as a whole.

And so what that leaves us with for, again, quantitative concentration response functions are these set of publications that do not have the arrows and the shading, all the ones that begin with Harvard Six Cities or ACS. ACS standing for American Cancer Society. And so that is the set of studies that we felt, and a similar conclusion has been arrived at by

EPA and the National Research Council and others, this is the set of studies that could let us develop a quantitative concentration response function. We then certainly proceed to look at the estimates from those studies and try to quantitatively synthesize them.

And so you can see here in the exposure increment column here, all the exposure increments here are 10 micrograms per cubic meter of $PM_{2.5}$ on an annual average basis. And in our report we provided concentration response functions for 1 microgram per cubic meter of $PM_{2.5}$. So our numbers will all be a factor of 10 lower than the numbers in this column. So everything is just scaling up and down.

But you can see that the central estimates, those are the numbers before the parentheses in this all cause column here. Those numbers representing the best estimates within those studies range in general from about 6 percent as the lowest number up to 17 percent as the highest number; or in the scale that we used, .6 percent up to 1.7 percent per microgram per cubic meter.

We looked at that and then looked at other evidence and other discussions of the two studies, weighted that and decided that a value basically part way in between what the different studies were providing was the most reasonable best estimate. So what we arrived at is a value of 1 percent per microgram per cubic meter, again, within that range between

the .6 percent and the 1.7 percent.

The last thing that I just want to highlight here briefly, and I know that this has been going through this table in some detail, is this row that -- here where there's an 8 to 11 number which I have conveniently obliterated with my circle, but what this is showing is some people had concern that the American Cancer Society study may not be perfectly representative of U.S. population because it's a higher educated population than in the U.S. as a whole and a number of studies have -- both these studies and the literature as a whole has shown that lower education people may be at greater risk of air pollution than higher education people related to socioeconomic status.

So calculations were made saying what if the American Cancer Society study had a population that had education like the Six Cities study which was more representative of the U.S, and that increased their numbers to something like 8 to 11 percent or .8 to 1.1 percent.

So that was one of many indications that the numbers -- the original numbers from the American Cancer Society study might be a little bit too low for a general U.S. application. So that helped to lead us to a value of 1 percent that we thought was well supported by this variety of studies.

Actually, the last point I want to mention -- I know my other point was also the last point -- is just to say each of

these cohort studies has multiple publications and that's because there's been a series of follow-on studies. The original publications. The studies were then reanalyzed by independent research teams. That's the papers by Krewski et al. And then there were what are called the extended analyses as more people in the cohorts died so there was more statistical power to see effect. So each of these cohorts has been analyzed in multiple published studies by different sets of authors.

Q. And has there also been a recent expert panel impaneled by the USEPA that looked at a similar mortality, premature mortality concentration response function related to $PM_{2.5}$ pollution?

A. Yes, there was. EPA launched into a few different efforts to better establish mortality concentration response functions. For ozone, as I mentioned, they funded myself and others to conduct meta-analyses of the literature.

For $PM_{2.5}$ what they did was engaged in a formal expert elicitation protocol to ask experts in the field what function they would consider the totality of the evidence to provide. And this is a very rigorous, lengthy, somewhat complex exercise. You know, it's not just calling 12 friends and asking them so what do you think. The experts were recruited in a very systematic and rigorous process. They received lengthy lists of publications, both these cohort studies, time

series studies, toxicologic studies. They went through a systematic and rigorous peer-reviewed approach to elicit their opinions to determine what they consider to be the best estimates from the literature as well as a plausible range of values. And finally arriving at essentially these best estimates and ranges across the 12 experts that could then be utilized by EPA in better understanding the literature and in developing a PM concentration response function for their future regulatory impact analyses.

Q. And did you include in your report, Dr. Levy, a summary of where the experts in the -- what they refer to as the expert elicitation, USEPA's expert elicitation where they came out on the concentration response function for premature mortality related to $PM_{2.5}$ emission?

A. Yes, we did.

Q. I'd like to show you Plaintiff's Exhibit 239 for identification. Could you identify this figure and tell us what it shows.

A. So this figure shows the --

Q. And if you would, Dr. Levy, I'm sorry to interrupt you, but you might be able to clear your screen.

A. How do I do that?

Q. By hitting one of the corners that's labeled clear.

A. Oh, there you go. Thank you.

So what this figure shows are the final opinions of the

12 experts impaneled in the study. That's experts labeled A through L, so the opinions are not matched to the individual so it's somewhat anonymized, although the 12 experts' names were identified in the report.

There are then the concentration response functions from two of the major cohort studies, Pope et al 2002 here. That's one of the publications from the ACS study. Dockery et al 1993. That's one of the publications from the Six Cities study.

And then the final column is showing the central estimate that we used in our calculations.

And so you can see that the expert values certainly range across the experts, but fall within a fairly narrow band. And you can see the vast majority of the central estimates which are represented by the black dots in these functions tend to fall between roughly 1.5 percent and .5 percent per microgram per cubic meter of $PM_{2.5}$. You can see one of the experts was a little bit higher than the rest at 2 percent. One of the experts was a little bit lower than the rest at around .4 percent. But that the full body of opinions really sits very close to a 1 percent value. And you can see that our value over here on the side falls right within the middle of the range of the values that these experts provided.

Q. And can you tell us what the -- what are they called, box-and-whisker components of this figure? Did I get that

right?

A. Yeah, that's correct. The dot in the middle represents the best estimate or, you know, the 50th percentile of the distribution. So this is part of the experts' uncertainty distribution around those values.

The two sides of the boxes here and here, roughly, are the 25th and 75th percentile of the distribution. So basically, you know, half of their distribution of plausibility for this function lies within that box.

And then the bars go from their 5th percentile to their 95th percentile. So these are sort of the broader bounding values.

So if we take, you know, expert, you know, E, as an example, he said that his best estimate was 2 percent, you know, that his sort of range of plausible values for the function from the 5th percentile to the 95th went from about 1 percent to 3 percent and then sort of the 25th to 75th percentile, sort of the meaty part of the distribution is in this box between, say, you know, 1.6 percent and 2.4 percent roughly.

Clear all that.

Q. And did EPA publish the results and the method of the expert elicitation in a formal report?

A. Yes, there was a report that was prepared by Industrial Economics and then was published by the EPA in, I believe,

September of 2006.
Q. Show you Plaintiff's Exhibit 242. Is that a true and correct copy of the expert elicitation report that was published publicly by USEPA?
A. Yes, it is.
Q. And that contains a description of the method that they used of selecting, impaneling this expert group and then having them review the literature and develop their assessment of concentration response functions for $PM_{2.5}$ exposure.
A. Yes. That's a very lengthy, detailed and rigorous methodology. You can see this is page 2 of 109. So they, you know, went through a very systematic approach to elicit these opinions, to develop these functions and to arrive at the values that could then be used in future health impact assessments.
Q. And can you just give us a short, couple minute overview of that process.
A. Sure. It's, you know, actually pretty well captured in a diagram that is labeled page 5 of 109 within the report. And yeah, it's up on the screen now.
And so you can see they went through a process, you know, first developing a protocol. A protocol meaning the means by which they elicited the opinions of the experts on these functions. That protocol was developed over a series of, I think, a year and a half or two years and involved pilot

applications, some pretesting, a symposium, gathering some reviews from a number of experts in the field, testing out the protocol both in this pilot phase and in a number of other settings.

They then went through the expert selection process which, as you can imagine, is a very important step in this sort of elicitation. They essentially looked at the peer-reviewed literature for publications over, I believe, the last 50 years. They found the 32 authors who had the largest publication record on the health effects of fine particulate matter, and then contacted them and asked them who do you think would be able to answer this question in a very clear and unbiased fashion. And there's a lengthy description of how they divided the 32 experts into four groups and asked them differently formulated questions to try to come up with those experts.

They then took the nominations, ranged them and started contacting people from the top of the list working their way down.

They then, since they wanted to make sure there was broad representation of scientific opinions, and the panel they had gotten to date through that process had largely epidemiologists, they asked the Health Effects Institute, which is a nonprofit entity jointly funded by EPA and industry, to come up with nominations of toxicologists or

other physicians not epidemiologists who could also be part of the panel. And they were contacted in a rank ordering approach, ending up with the 12 experts.

You can see there's -- the next step in this flow chart, briefing book development. This involved pulling together hundreds of publications in the literature so that all of the authors -- I'm sorry, all the experts had all studies available to them that they could reference. I think there was also a laptop computer present when they were doing their elicitation so any study could be pulled up at any point in time that they could review.

There was a workshop that the experts attended so they could learn about potential biases in people's opinions, what expert elicitation involves, trying to make sure that you're a well calibrated expert -- meaning that, you know, when you provide these uncertainty bounds, you're not wildly too large or wildly too small in those bounds -- as well as discussing what the evidence looked like, what the literature was and letting the experts sort of hash through the nature of the evidence.

They went through the interviews which were eight hour interviews, full day, at the expert's office going through these protocols. They had real-time computer interface so that at each step the expert, you know, gave an opinion, they could immediately tell them what that would mean for their

quantitative function.

And it's a two person interviewing team, one of whom is meant to be -- I'm sure there's a better term for it, but the argumentative person. So if the expert gives a high number, the argumentative person points out the evidence that could lead to lower estimates. If they give a lower number, they point out evidence that could lead to higher estimates and force them to confront any issues in the literature.

They then had a post-elicitation workshop in which the experts discussed the entire approach, gave their opinions about it and then allowed the experts to, based on further discussion of the literature, change any of their opinions if at that stage they felt it needed to be changed. There were actually very, very few changes at that stage, but a couple of experts made minor changes.

So it was a lengthy process. It spanned a couple of years. Involved, you know, many people in very rigorously developed protocols.

Q. And the final expert list of the experts that were selected and participated on the panel, are they on Page 6 of 109, the next page of this exhibit?

A. Yes, they are.

Q. And do you know these experts?

A. I know some personally. I know others by reputation. But it certainly is a group of people who have thought about

this problem a lot and have published on it quite extensively.

MR. GOODSTEIN: Can we blow that up on the screen, please.

THE WITNESS: We've got it up here.

Q. And we noticed, Dr. Levy, that on the figure you showed before, Plaintiff's Exhibit 239, that the experts were listed by letter. Can you tell us how that worked.

A. Well, everyone was assigned a letter just to make sure that the opinion could not be tied to an individual expert so that that could let them be essentially free to express their opinions so the world would not know precisely what they had stated. You know, it allowed them to, you know, really be more open about their opinions on the literature.

Q. So this list of experts on Page 6 of Plaintiff's Exhibit 242 are the experts that you list as A through L on your summary, Plaintiff's Exhibit 239.

A. That's correct.

Q. And can we go back to Plaintiff's Exhibit 239 for a moment.

So as you explained to us earlier, each of those experts selected a concentration response function for $PM_{2.5}$ mortality that was consistent with the one you and Dr. Spengler selected.

A. That's correct. I think another thing to point out, which is on the heading of this figure, is that, you know, the

experts were not just asked one question about, you know, what are the effects of PM mortality. There was a series of questions, including whether there were thresholds for the effects and at what levels of ambient pollution those effects were observed. These are obviously critical questions from the perspective of EPA in developing their future health impact assessments.

So you can see the heading says these are the coefficients at a baseline annual average $PM_{2.5}$ level of 7 micrograms per cubic meter. So these were -- they asked the experts what functions they thought would be applicable at a level of 18 micrograms per cubic meter and they then asked them what functions are applicable at 7.

Furthermore, they then let the experts put any shape on the function that they wanted to put on it, you know, setting a threshold anywhere they would like to set it, creating non-linearities in the function if they wished to create them.

But what this figure shows extracted from the expert elicitation report with our opinion appended on at the right-hand side, shows the values that the experts gave at an ambient level of 7 micrograms per cubic meter on an annual average basis.

Q. So how would you describe the relationship of your concentration response function that you used for this case and the concentration response functions derived by these

other experts?
A. I think it's certainly very consistent. I think there's some experts who believe that we understated the effects. There's some experts who believe we overstated the effects. But I think that the value that we've arrived at is in the center of the values that are provided here and provides a best central estimate for health impact assessment.
MR. GOODSTEIN: Your Honor, at this time I offer Plaintiff's Exhibits 225, 226, 239 and 242 into evidence.
THE COURT: Let those be admitted.
(Plaintiff's Exhibits Numbers 225, 226, 239 and 242 were received into evidence.)
Q. All right, Dr. Levy. We'd like to walk through your results now. And let's start with -- oh, we've got one more background piece, I'm sorry. We wanted to have you explain a little bit, Dr. Levy, about how you link the air quality modeling results to the population and -- population affected, and this would be the last box in your analysis that we didn't cover. Apologize. The risk characterization for the analysis.
A. Yes. So as mentioned in the box, the exposure assessment and dose response steps get us to the percentage change in health outcomes that would be associated with the excess emissions from the TVA facilities. We then need to understand how many people are exposed to these excess concentrations or

excess changes in health effects and what the baseline rates of those effects are. The total health impacts are a product or a multiple of the change in concentration, the concentration response function, the number of people who are exposed and their baseline rate of disease or of premature death.

So for population data, we took data from the U.S. Census for the year 2000. We took those data at the Census tract level which is a very small level of geographic aggregation, averages about 4,000 people. And then linked those data up with the concentration model conducted with CMAQ which was at a different level of spatial resolution. So we used Geographic Information Systems software to overlay one on the other and calculate it on a similar spatial scale.

And then we used publicly available databases to get at the baseline rates of death or disease. For example, for premature death, there's a database that CDC, Centers for Disease Control, maintains called CDC Wonder and that has mortality data by county across the United States. And so we used those historic data.

For other health outcomes, the data sources varied but are described in our report and include different CDC databases of rates of hospital admissions by region, for example, or of emergency room visits by region.

Q. All right. And do you have an example in your reports of

the method you used to link the air quality modeling to the population information?

A. Yes, I do.

Q. Referring you to Plaintiff's Exhibit 227 for identification. Can you describe for us what this shows.

A. This shows the lines -- the grid lines correspond to the grid cells from the CMAQ model conducted by Mr. Chinkin and Mr. Wheeler. The other boundary lines here correspond to Census tracts here within North Carolina and then blown up here around the Asheville area. This shows that Census tracts overlap the grid cells within the CMAQ model. So we had to figure out, for example, what fraction of the population in this specific Census tract was located within each of the grid cells.

So by doing this GIS overlay, we could say 42 percent of the population is found here within this grid cell, 8 percent of the population found in this grid cell, and so forth, and so we could figure out, you know, by doing this for every single Census tract how many people live within each of the grid cells modeled by Chinkin and Wheeler.

Q. All right. So you talk about the baseline incidences of these health outcomes that you used. Can you describe for us how you combined these inputs to estimate the impacts on these health endpoints associated with TVA's emissions and the benefits associated with the additional controls.

A. So it was just a direct calculation of the excess concentrations associated with those emissions for $PM_{2.5}$ and ozone, in each case using the averaging time appropriate for the health outcome being considered and the concentration response functions. We linked that to the concentration response functions that we derived, and then with the population data in each case in the appropriate age bin for the health outcome. So for example, if the study of cohort mortality only looked at adults age 30 and over, we only considered those age 30 and over to be at risk for the health outcome so that we're consistent with the epidemiologic literature.

So within each of the cells we multiply it by the number of people in the appropriate age group and then by their baseline mortality rate or baseline incidence rate of various health outcomes. And so it's just the multiplication of those four values within each cell and then aggregated up across states and across the region.

Q. Did you also look at population projections for 2013 in addition to the 2000 Census population?

A. We did. For most of our calculations we relied on 2000 population which we know obviously will systematically underestimate the number of people living in the United States in 2013, but this was based on direct Census data and represented, you know, good measured data with, you know,

essentially no uncertainty.

We also did calculate what the effects would be for 2013 projected population data. There's a company called Woods and Poole who develops population projections for many purposes, USEPA, regulatory impact analyses among those purposes. So relied on those Woods and Poole population projections for some of our calculations.

Q. All right. And you presented your results both in a summary fashion and also state by state?

A. That's correct.

Q. So let's walk through your results, then, Dr. Levy, out of your reports starting with Plaintiff's Exhibit 228 for identification.

MR. LANCASTER: Your Honor, I need to interpose an objection. This exhibit tallies up the premature mortalities and other health endpoint quantifications of Dr. Levy, not just for North Carolina but for a, I believe it's a 33 state area and includes matters which, as I understand the court's rulings, are outside what's at issue here.

MR. GOODSTEIN: Your Honor, the testimony has been clear that this is the appropriate health impact assessment methodology to use. Both sides have modeled the CMAQ modeling domain and looked at the impacts of these sources on regional air pollution, including North Carolina. North Carolina's claimed in this case from day one that there are impacts

occurring in North Carolina from pollution from TVA power plants. They've also alleged that there's impacts on regional air quality.

And we understand the court is not interested in us focusing in on detailed impacts on states throughout the region. But what we've got here is a standard application of the health impact assessment methodology that this court has found to be reliable. And Dr. Levy's going to walk through these results in a couple of minutes here showing the totals for the region, also showing the state-by-state results so that the court can consider these results however it deems appropriate.

THE COURT: I already indicated that that would be the general approach and that the primary results that I'm considering are the four states involved in this lawsuit.

MR. GOODSTEIN: All right, Your Honor. And as we laid out -- if I might take another minute at this point since this is an important issue for the State of North Carolina.

We responded in detail to the motion in limine that was filed by Tennessee Valley Authority on this. They want to take the position that the only thing that this court should consider in this case is the impacts on air quality in North Carolina. This motion, as we laid out in our papers, Your Honor, was late. There was a deadline in the Case Management Order of February of this year for any motions in limine

related to the scientific evidence. So it was an untimely motion.

This case has been litigated the way it was pled in the complaint dealing with impacts to air quality in North Carolina and the region and that's the way we did the discovery in the case. That's the way we did the expert reports in the case. That's the way we prepared for trial in this case, Your Honor.

THE COURT: We've been through that, I think, Mr. Goodstein.

MR. GOODSTEIN: Your Honor, if I could just take one more minute of the court's time.

THE COURT: Go ahead.

MR. GOODSTEIN: This is -- this issue of impacts to air quality throughout the region, while we're not going to spend very much more time on it at all, we want the court to know that is relevant to both liability in this case and injunctive relief. The court has found that the Restatement of Torts and the explication of public nuisance law in the Restatement as well as the law of the source states allows this court to consider the impacts not only to the plaintiff in this case but to the general public. That's the essence of public nuisance. And now that this court has found that North Carolina has standing here, Your Honor, it's the state's position that the court should look at the benefits to the

public as well as the benefits to North Carolina.

THE COURT: I understand and I've heard your point now on several occasions.

MR. GOODSTEIN: Thank you, Your Honor.

THE COURT: And you may look at my decision as to how and what I considered to be appropriate in this case.

MR. GOODSTEIN: Thank you, Your Honor.

THE COURT: Yes, sir.

BY MR. GOODSTEIN:

Q. All right. Dr. Levy, can you tell us what Plaintiff's Exhibit 228 shows.

A. Yes. This shows our best estimate using 2000 population data of the reduction in mortality and morbidity across the region that would be associated with the reductions that were described by Dr. Staudt and then modeled by Messrs. Chinkin and Wheeler. This includes -- these are annual reductions so benefits that would accrue each year.

This includes 1400 fewer premature deaths. I won't read each of the outcomes here to save the court's time, but you can see a variety of morbidity outcomes that we've calculated, hospital admissions for respiratory or cardiovascular disease, emergency room visits for asthma, as well as asthma exacerbations or asthma attacks, development of cases of chronic bronchitis, minor restricted activity days and school loss days.

Q. All right. And can you now turn to Plaintiff's Exhibit 243 for identification and I ask you to explain what that figure shows.

A. This is the identical table to the prior one, the same health outcomes, the same framework. The only difference is this is using the projected population for 2013 as conducted by Woods and Poole. You can see as expected the benefits increase. There's more people who are living in the region and at risk. The benefits increase by roughly 13 percent for premature death as well as the morbidity outcomes.

Q. All right. And Plaintiff's Exhibit 229 for identification.

A. We're now going back to the 2000 population, and all the remaining figures that we had are based off of 2000 population, so keep that in mind.

And this is looking at the benefits only associated with fine particulate matter. So this is dividing the total benefits from two figures ago into the damages from fine particulate matter and then later the damages associated with ozone specifically.

Q. All right. And Plaintiff's Exhibit 230 for identification.

A. This is now the benefits associated solely with ozone in the region, not including fine particulate matter, based on 2000 population data. You can see that there are fewer health

outcomes listed because some of the outcomes within our table were solely attributable to fine particulate matter and not to ozone, but the benefits included in this case premature mortality as well as various morbidity outcomes.

Q. And Plaintiff's Exhibit 233 for identification.

A. This exhibit is a map from our report focusing on the spatial distribution of the benefits. This is based off of 2000 population data so these numbers, if you can add very quickly, would sum up to the 1400 value I described before.

This is premature deaths averted. You can see that within Tennessee, that has the highest number of premature deaths averted at 180 per year, approximately 99 within North Carolina, 891 in Kentucky, 77 in Alabama, and so forth.

Q. And Plaintiff's Exhibit 235 for identification, can you please explain what that one shows.

A. This is a similar map of the spatial distribution of benefits, in this case focusing on hospital admissions. This is cardiovascular plus respiratory hospital admission, so adding up those two rows in the table. And again, how those benefits are distributed across the states based on 2000 population data. Again, greatest benefit exhibited in Tennessee at 210 per year, North Carolina at 120 per year, and so on.

Q. Plaintiff's Exhibit 236 for identification.

A. We're now looking at the outcome of lost school days.

Everything else is identical to previously, 2000 population data utilized. I'm looking at how those benefits are distributed across the states. Again, to sound like a broken record, the greatest benefits exhibited in Tennessee at 7200 lost school days averted per year, 2300 lost school days averted among children in North Carolina per year.

Q. And Plaintiff's Exhibit 231 for identification, what does that show?

A. This is a table of the benefits solely occurring within North Carolina. You can see in the top row is the 99 that we saw on the map a few figures ago and so this is based on 2000 population. The benefits anticipated within the state of North Carolina from the controls on the TVA facilities: 99 fewer premature deaths per year, 19,000 fewer asthma exacerbations per year, and so forth as exhibited in the table.

Q. And for clarification, these are per year impacts and benefits.

A. Yes, they are annual benefits.

Q. So for each year that the emissions are reduced from TVA power plants as requested by North Carolina, would you expect this type of health benefit to accrue in North Carolina and in the other states that you've summarized?

A. Yes, I would. And to the extent that we used 2000 population data and then are using that for all years going

forward, the benefits would certainly be greater in subsequent years as the population continued to grow. So this would be a somewhat conservative estimate of what those benefits would be over time.

Q. And Plaintiff's Exhibit 232 for identification, can you explain what that shows.

A. This is now dividing out the component of the previous table focused solely on fine particulate matter. So this is the portion of the benefits from the controls on the TVA facilities found in North Carolina just related to fine particulate matter. So 98 premature deaths avoided per year, 42,000 minor restricted activity days avoided per year, and so forth in between.

Q. Plaintiff's Exhibit 234 for identification, can you explain what that shows.

A. This shows everything in short-term. I will not talk about these numbers, but this is the impact broken out by state and by health outcome for every state and every outcome considered in the analysis. And I should say the states are ordered by the magnitude of the impact on premature mortality. So largest impact down to smallest impact.

Q. And Plaintiff's Exhibit 237 for identification.

A. This is now essentially a blown up version of the prior table focusing just on premature deaths, and again, sorted from high to low. Identical calculation as presented in the

previous table year 2000 population base.

Q. And Plaintiff's Exhibit 240 for identification, can you explain what that one shows.

A. This is a calculation that requires a little bit of an explanation. Certainly one of the things within our assessment that we had to consider was the shape of the concentration response function and whether a threshold was exhibited. There's certainly a variety of assumptions that could be made within these calculations. We felt that the epidemiologic literature showed that effects were exhibited down to the lowest levels found within the region. But we wanted to understand if we had assumed a threshold to be present for effects, a threshold that I believe was suggested by Dr. Anderson if I'm correct, what would the implication be for our calculations?

And so this, I should say, should be considered as a decidedly lower bound calculation. I think the scientific evidence first shows that there are effects below 10 micrograms per cubic meter. There's also, within EPA practice, when health impact assessments are conducted, if a threshold is put into place when the evidence seems to indicate a linear relationship, they use what's called a hockey stick dose response function which basically is a flat line out to the threshold, which in this case was 10, and then a slope increasing upward from there. And if you use a hockey

stick dose response, the slope on the upward part of the hockey stick should be considered as higher than the linear slope.

We didn't use a higher slope in this case which is -- the higher slope is the practice EPA would utilize. So we're using what we would consider a threshold not supported by the literature and not making that upward adjustment. And this shows that of our mortality and morbidity benefits calculated, approximately 70 percent of them would occur in this region above 10 micrograms per cubic meter. Again, I would consider that 70 percent to be an extreme lower bound calculation that I don't feel is well supported by the evidence.

Q. And we'll talk about the issue of a threshold and whether there is any evidence of one in a minute, but this is basically an exhibit that responded to some issues that were raised by Dr. Anderson and Dr. Moolgavkar.

A. That's correct. It was taking their statements at face value and what the implications of those would be.

MR. GOODSTEIN: So I want to offer, Your Honor, the results tables that Dr. Levy and Dr. Spengler included in their reports and that we just went over. These are Plaintiff's Exhibits 228, 243, 229, 230, 233, 235, 236, 231, 232, 234 and 237 into evidence at this time.

THE COURT: All right. Let them be admitted.

MR. LANCASTER: May I go ahead and note my objection

for the record, sir.

THE COURT: I thought you already had.

MR. LANCASTER: Okay. I didn't know if I needed to do it again. Thank you.

THE COURT: Certainly your objection is now on the record.

MR. LANCASTER: Thank you, sir.

(Plaintiff's Exhibits Numbers 228, 229 230, 231, 232, 233, 234, 235, 236, 237 and 243 were received into evidence.)

Q. Dr. Levy, with regard to the results that we just went through, based on your experience, are these estimates of the number of avoided health outcomes and impacts on health -- public health from TVA's current emissions, are these reasonable and reliable estimates?

A. Yes, they are. As we described, they relied on standard practice within health impact assessment, utilized the state-of-the-art atmospheric dispersion model to estimate exposures, relied on what a very large epidemiologic literature as well as the corroboratory toxicologic literature would say are the best concentration response functions, and then relied on very standardized population databases in a very straightforward calculation approach.

And so I think each of the steps represents best practice in the field and the values that we've presented represent

what we would consider to be reasonable best estimates of the benefits of controls.

Q. And you've had an opportunity to review TVA's expert reports in this matter, in particular the reports of Dr. Moolgavkar and Dr. Anderson.

A. Yes, I have.

Q. And did any of their comments on your analysis change your conclusions?

A. No, they did not.

Q. In particular, I want to ask you about a few issues. We talked earlier about the issue of whether or not there's any scientific evidence of a threshold below which there are no human health effects for either ozone or $PM_{2.5}$. Do you recall the portions of Dr. Anderson and Dr. Moolgavkar's reports that dealt with this issue?

A. Yes, I do.

Q. Can you explain to us why those portions of their reports didn't change your conclusion or Dr. Spengler's conclusion in any way.

A. Well, to determine whether there is or is not a threshold for these pollutants and effects, we rely on the empirical evidence, and so we look, for example, at the concentration response functions that have been shown in studies like the Six Cities study, the American Cancer Society study and other published studies. And what those studies have shown are

functions that are quite linear down to the lowest concentrations observed within those studies. The most recent publications from the Six Cities study were showing effects down to 7 micrograms per cubic meter on an annual average basis.

And so these relationships are very consistent. They're reasonable. And on its face it did not seem plausible to think that within the range of values that happened to occur within the epidemiologic studies, there was a strong significant linear relationship that would then hit a brick wall and stop precisely at the lowest value that happened to be measured. So that did not seem like a plausible interpretation of the literature, and time series studies as well as cohort studies have demonstrated effects down to very low levels.

So we felt that the empirical evidence did not support their conclusions as well as even a theoretical consideration of the likelihood of a threshold in this case.

Q. Do you believe that the NAAQS, the National Ambient Air Quality Standards, are an appropriate threshold below which health effects from exposure to $PM_{2.5}$ or ozone do not occur?

A. No, it is not such a threshold. EPA has articulated on many occasions that their NAAQS is not meant to be a zero risk level. When EPA conducts its health impact assessments, it uses concentration response functions and quantifies benefits

below the National Ambient Air Quality Standards. The empirical evidence strongly supports the existence of health effects below the current National Ambient Air Quality Standards.

So I think it is quite clear that either on a theoretical or empirical basis the NAAQS should not be considered as a population threshold.

Q. Do you agree with the use of a threshold at all in a health impact assessment like the one you and Dr. Spengler performed in this case?

A. As stated, any threshold would have to be supported by empirical evidence and the evidence does not indicate a threshold down to the lowest levels that have been observed within these cohort studies. My interpretation would be that a linear function down to the lowest levels within our domain is then very well supported by the literature.

You know, whether a threshold exists at levels that are not exhibited anywhere within this domain in 2013 and have not yet been investigated, you know, is an interesting theoretical question, but it is not a relevant question for our application. You know, the question is are health effects exhibited in the range of concentrations that were projected to occur in 2013 within this domain? And the empirical evidence strongly supports a linear concentration response function without a threshold in that range.

Q. Did the experts, the 12 experts in EPA's recent expert elicitation, did they provide opinions on the existence of a threshold?

A. They did. As mentioned, this was part of the elicitation to let the experts set thresholds wherever they thought was appropriate and to look at any non-linearities in the functions as a result.

I believe 11 of the 12 experts said there was no indication that a threshold would exist either on a theoretical or an empirical basis. The twelfth expert thought that there could be a threshold. I believe he said there was a 50 percent chance that there was and 50 percent chance that there wasn't. And if there was threshold, that it would most likely be exhibited below 5 micrograms per cubic meter, and with certainty at less that 10 micrograms per cubic meter.

So looking at the totality of the literature and thinking about the biologic plausibility for a threshold as well as the empirical evidence for one, it unanimously concluded that effects would be exhibited above 10 and nearly unanimously concluded that effects would be exhibited above 5 with one small probability placed on a threshold between 5 and 10.

Q. And 11 out of 12 experts, what did they conclude about whether a threshold was consistent with the literature?

A. They concluded that it was not consistent with the literature.

Q. Dr. Anderson, one of TVA's experts in this case, she has suggested in her reports the use of an assumed threshold of 10 micrograms per meter cubed and we alluded to this earlier.

MR. LANCASTER: Your Honor, I object. Of course, the evidence will speak for itself, but that wasn't Dr. Anderson's statement in her report. I believe he's got Dr. Anderson confused with Dr. Smith.

MR. GOODSTEIN: I'll rephrase, Your Honor.

THE COURT: All right.

MR. GOODSTEIN: Thank you.

Q. Are you familiar with the position espoused by at least one of TVA's experts that there may be -- it may be appropriate to assume a threshold of 10 micrograms per meter cubed?

A. I'm familiar with that.

Q. Do you agree with that approach?

A. I do not. I think, you know, 10 is a number that, frankly, I didn't see any rationale for or basis for. It's a number as good as any other number, but there's no rationale for a threshold there. There's not been one study that has demonstrated a threshold at 10 micrograms per cubic meter. Studies have exhibited effects below that level, so I don't think there's any rationale for that value.

Q. And are there studies published in peer-reviewed literature that show effects from concentrations of $PM_{2.5}$

below 10 micrograms per cubic meter?
A. Yes, there are.
Q. And could you describe those for us, please.
A. The most recent publication was an article by Schwartz and colleagues in the journal Environmental Health Perspectives, and this article was interesting from a few perspectives. I mean, one, it included lower concentrations. It was a more extended follow-up of the Harvard Six Cities study and so it had concentrations down to 7 micrograms per cubic meter on an annual average basis.

The study also explicitly looked for thresholds and so Dr. Schwartz and colleagues took 32 possible shapes of concentration response functions with thresholds at different points and then looked at how those thresholds were supported or not supported by the empirical evidence and used sort of statistical means to determine what shape of the concentration response function was best supported. And they concluded that far and away the most supported shape is a straight line all the way from 7 micrograms per cubic meter up to the highest level, and that there was, you know, no empirical evidence that was supportive of, you know, a hockey stick shape or a threshold below 10 or below any of the levels observed within the Harvard Six Cities study.
Q. Can you tell us about the recent California Air Resources Board report that you testified yesterday that you peer

reviewed.

A. Yes. CARB, or California Air Resources Board, has been developing methods for health impact assessment in a variety of contexts. I think currently looking at their goods movement initiative, trying to understand the effects of moving goods around ports and roads in California. And so they wanted to develop an appropriate concentration response function for PM mortality as a component of that. So they looked through the literature, prepared a report, had that report reviewed by a number of experts.

And I think their key conclusions were, first, they determined that an appropriate best estimate for the PM mortality concentration response function was a 1 percent increase per microgram per cubic meter of annual average $PM_{2.5}$, so the same function that we determined.

They relied on the evidence from the expert elicitation and considered that to be a very valuable and reliable resource. And they explicitly addressed the question of possible thresholds or what they termed as cutoff points. They felt that there was no evidence for a threshold, but they wanted to look at the effect at a series of what they deemed cutoff points. They thought that the highest cutoff point that was supported by the evidence was 7 micrograms per cubic meter on an annual average basis, and then they looked at other cutoff points down to nonanthropogenic background levels

in California.

And so basically said, you know, this function of 1 percent is very clearly indicated for levels of 7 micrograms per cubic meter and higher. It seems likely to be indicated at lower ranges, and they tested the sensitivity to a few of the alternative possible cutoff points.

Q. Have you also reviewed TVA's experts' assertion that a sigmoid curve is appropriate for population level health effects?

A. Yes, I have.

Q. And can you explain to us what that means and whether you agree with that proposition.

A. I agree with it in principle, but in this application it does not hold up. And if I may use another sheet on the board there.

MR. GOODSTEIN: With Your Honor's permission, may Dr. Levy approach the pad?

THE COURT: Yes.

MR. GOODSTEIN: Thank you, Your Honor.

(Witness stepped down from the witness stand.)

THE WITNESS: So I'll try to be brief, but, you know, just to understand what a dose response curve is and what sigmoid is, it's helpful to sketch it out.

So it's very well accepted by toxicologists and by most people in the human health field that as individuals, we

exhibit thresholds for pollutants. So for myself, there is a level below which I will not be affected by fine particulate matter or ozone. So if we draw my dose response curve, me as an individual, it will look like this -- those are my key flat lines -- where basically nothing happens for a while because my body can repair the effects. Then there's a level at which I exhibit an effect and so this is sort of having an effect up here and this is having no effect. So it has this sort of step look to it. So this is very commonly accepted and this is what one of TVA's experts referred to.

Now, one of the issues is that we are very heterogeneous. Humans differ in their suseptibility and their sensitivity so my curve might look like that. Someone else in this courtroom's curve might look like this. Another person's may look like that. So we all as individuals have different thresholds. And when you add up those different distributions of thresholds, what the curve usually looks like is this sort of S-shaped function or the sigmoid function that was described by one of TVA's experts.

But the question is how steep or how flat is this S shape. If everyone was identical, it would be very steep and it would basically look like one of these curves. It would be totally flat, then it would jump up and then it would be flat.

And when you look at an animal study with a number of rats that are generally inbred within a specific strain, they

exhibit some variability in their suseptibility, but not as much. They're inbred rats. So their curves look in this very S-shaped form.

Now, if you have humans who differ in their genetic profiles, in whether they smoke or not, in whether they're obese or not, in their exposures to air pollution, a number of other effects, the S shape flattens out.

And so this is a curve where people don't differ that much in their susceptibility.

This would be a curve where people start to differ more in their susceptibility.

And if people differ a lot in their susceptibility, it looks like a straight line.

And for the kinds of health outcomes we're concerned with here, say cardiovascular disease, we know there's a lot of genetic factors, dietary factors, smoking status, all sorts of other things that can predispose one to cardiovascular disease. Humans are very, very heterogeneous creatures. So we would exhibit this sort of a shape in principle if we didn't differ. But in fact, we look more like this because we're much more heterogeneous.

And so a linear dose response function for these pollutants is very consistent with what we know about human sensitivity and it's also very consistent with the theoretical structure that TVA's experts laid out, but not with the fact

that we're not inbred rats.

(Witness resumed the witness stand.)

MR. GOODSTEIN: Your Honor, we offer Plaintiff's Exhibit 486 into evidence which is Dr. Levy's sketch.

THE COURT: Let it be admitted.

(Plaintiff's Exhibit Number 486 was received into evidence.)

Q. I want to turn to nondifferential toxicity, which is just what I'm using to characterize some of the comments in the TVA expert reports about what's an appropriate weight of toxicity for the constituents of $PM_{2.5}$, and where would particles contributed by emissions from TVA's coal-fired power plants, where would they fall in that spectrum of toxicity. Is this an issue that you considered in your analysis with Dr. Spengler in this case?

A. Yes, it is.

Q. And you're familiar with the comments made by the TVA experts on this issue.

A. Yes, I am.

Q. Can you explain to us what appropriate consideration you gave to this argument of differential toxicity between components of $PM_{2.5}$.

A. It's certainly a complex topic there's a lot of ongoing scientific research on, you know, but EPA, as one example, has clearly indicated that there's no evidence at present that

would allow specific constituents of $PM_{2.5}$ to be exonerated. I think that has been clearly stated. And within all their health impact assessments, they further said there's no evidence that would allow us to quantitatively deviate from that base assumption of equal toxicity.

So our conclusion in this matter is very similar to -- in fact, identical to EPA's conclusion and practices.

Moreover, we have examined the epidemiologic literature and toxicologic literature for sulfate and nitrate particles. The literature is far larger on sulfate particles than on nitrate particles, but especially for sulfate particles there is good evidence of effects that are comparable to the effects to fine particulate matter.

The two cohort studies upon which we relied, the Six Cities study and the American Cancer Society study, showed concentration responses to sulfate particles that were higher than those for $PM_{2.5}$ as a whole.

There are a number of time series studies that have found effects of sulfate comparable to or greater than $PM_{2.5}$ as a whole.

There's also a number of studies of nonclinical -- I'm sorry, nonlethal endpoints that have been -- or preclinical endpoints that have been considered in the epidemiologic literature. Studies looking at heart rate variability, for example, which Dr. Peden described yesterday as an important

step in the causal pathway for cardiovascular effect. So there have been multiple publications that have shown sulfate particles attributed to changes in heart rate variability.

So, you know, looking at that totality of evidence, looking at EPA's conclusions on this matter, we felt that using identical toxicity for the different constituents was well supported by the evidence.

Q. And if you had to assign weight to the toxicity of constituents of $PM_{2.5}$, where would the $PM_{2.5}$ particles from TVA's coal-fired power plants fall in a range of toxicity?

A. It's certainly a tough question to do quantitatively. You know, one thing that one could do, for example -- you know, a lot of the attention has been focused on PM mortality since that's one of the more eye catching impacts. If we had applied the concentration response functions reported by the authors for sulfate for mortality, our impacts would have gone up somewhat. We didn't feel that that was necessarily the most appropriate thing to do. We wanted to be somewhat conservative in that regard.

So we used a value of identical toxicity, but relying on the empirical evidence from that study as well as from some of the published time series studies would have led to a slightly higher estimate for sulfate particles than for $PM_{2.5}$ as a whole. I don't think I'd be prepared, and I think the scientific community would agree, be prepared to assign

specific numeric values to a plausible range.

But I think any of the health impact assessments that I've observed both by USEPA, in Europe, and in the peer-reviewed literature have all used equal toxicity as the base assumption and none have exonerated species such as sulfates and nitrates which comprise a significant amount of the fine particulate fraction in certainly most parts of the United States.

Q. And is there some evidence that sulfates derived from fossil fuel combustion are more toxic than noncombustion particles?

A. There's certainly the studies I alluded to looking at sulfate and $PM_{2.5}$. There have been other studies on paper by Laden and colleagues in 2000 that used factor analysis methods to basically look at where the $PM_{2.5}$ was coming from and come up with concentration response functions for PM from motor vehicles, from coal combustion, and so forth. And so they found a function for coal that was lower than that for motor vehicles, but roughly comparable to or I believe slightly higher than that of the PM mass as a whole. And there's other studies that have similar corroboratory relationships. As I mentioned, the time series literature which on average shows higher concentration response functions for sulfate versus $PM_{2.5}$.

Q. And you're familiar with the TVA expert reports that

suggested that you and Dr. Spengler did not adequately consider uncertainty in your analysis in this case.

A. I'm familiar, yes.

Q. Did that number of comments about uncertainty cause you to reconsider your analysis here?

A. No, it did not. I mean, I think clearly, you know, as I stated at the outset, you know, uncertainty is a key component in risk assessment. It's the reason why we do risk assessment to shed light on problems where there is scientific uncertainty. And so we considered uncertainty along many dimensions. We explicitly talked about uncertainty in sections of our report.

Just as a couple of examples, you know, as we develop the concentration response functions for individual pollutants and outcomes, the statistical method that we used to pool the studies takes account of the uncertainty reported in the individual studies and weighs the studies according to their uncertainty so the more uncertain studies get less weight.

So that's a formal part of our statistical process for PM mortality which is a somewhat more complicated issue given that there's many publications from the same cohorts, so simply pooling them together in a quantitative analysis isn't necessarily the right way to go.

We looked at the evidence. We explicitly described the range of values that one could see across the different cohort

studies, as I mentioned earlier, from .6 percent to 1.7 percent. And we explicitly described in our report that if you considered the low end of expert opinions and published studies up to the high end of expert opinions and published studies, it might increase or decrease our mortality estimate by a factor of 2 in either direction. So in other words, our base value of 1400 premature deaths per year could be as high as 2800 if you rely on certain sets of evidence, could be as low as 700 if you rely on other sets of evidence. But we felt that our value of 1400 was the best available science, the best available evidence and that that would provide the best information to the court in trying to evaluate the evidence and come to some determination.

Q. So in conclusion, Dr. Levy, how would you describe the values for health impacts that you have associated with TVA's current excess emissions?

A. I would describe the values as representing best available science and best available practice and indicating that the current emissions do provide a substantial public health burden throughout the region, both in terms of mortality effects and morbidity effects for an array of cardiovascular and respiratory diseases.

Q. And how would you describe the health benefits that you have associated with the emissions reductions on TVA power plants sought by North Carolina in this case?

A. I would similarly describe our quantitative values as what the best available science indicates would be the case and that those benefits that would accrue due to the controls are substantial across the region on an annualized basis. And by substantial, I mean in comparison to the benefits that we found previously in modeling the benefits of power plant controls and for other sets of power plants in different areas of the country, and relatively large in respect to some of the national control strategies such as the Clean Air Interstate Rule that EPA had evaluated and considered.

MR. GOODSTEIN: Your Honor, if I could have a moment.

THE COURT: All right.

(Co-counsel conferred.)

MR. GOODSTEIN: Your Honor, I think I neglected to offer Plaintiff's Exhibit 227 for identification into evidence and would so offer that at this time.

THE COURT: All right. Let that be admitted.

(Plaintiff's Exhibit Number 227 was received into evidence.)

MR. GOODSTEIN: We have no further questions of Dr. Levy at this time, Your Honor.

THE COURT: All right. We'll take our midmorning recess of 15 minutes and then we'll begin cross.

MR. LANCASTER: Thank you.

(Brief recess at 10:53 a.m.)

THE COURT: Come back around.

(Witness resumed the witness stand.)

THE COURT: Mr. Lancaster.

MR. LANCASTER: Your Honor, if I may suggest that the witness and the court pick up TVA Book 15.

And I'd also request permission to approach the witness and the bench with a small notebook I have containing a number of articles that Dr. Levy has published in case he wishes to refer to them.

THE COURT: All right, sir.

JONATHAN LEVY

CROSS EXAMINATION

BY MR. LANCASTER:

Q. Dr. Levy, good morning.

A. Good morning.

Q. The calculations you made of health endpoints such as premature mortalities avoided, those calculations depended on inputs of information from others of the plaintiff's witnesses, correct?

A. That's correct, for the exposure component.

Q. For example, Dr. Staudt specified the TVA emissions assumptions both for the 2013 base case and the 2013 controls case, correct?

A. Correct.

Q. And you have not independently confirmed the accuracy of either the base case or the controls case that Dr. Staudt specified, have you?
A. That's correct.
Q. And you're relying on Dr. Staudt for those emissions endpoints, correct?
A. Correct.
Q. And then as we learned over the next couple days after Dr. Staudt's testimony, those emissions scenarios were run through air dispersion modeling by Mr. Wheeler and Mr. Chinkin, correct?
A. Correct.
Q. And you accepted the output of that modeling done by Mr. Wheeler and Mr. Chinkin, correct?
A. Correct.
Q. You did not perform the air dispersion modeling.
A. No, we did not.
Q. And you have not independently verified the air dispersion modeling performed by Mr. Chinkin and Mr. Wheeler.
A. We did some quality assurance checks on the outputs, but we did not obviously verify the full runs.
Q. One of the health endpoints that you calculated was premature mortalities avoided based on the modeling output that showed lower levels of $PM_{2.5}$ in the air if the emissions were at Staudt's controls case as compared to Staudt's base

case, correct?
A. Correct.
Q. On a county-by-county basis, you subtracted the $PM_{2.5}$ concentrations in the county, you subtracted the lower levels under the base case from the higher levels under the -- excuse me. You subtracted the lower levels under the controls case from the higher levels under the base case to get the concentration difference in each county, correct?
A. Correct.
Q. And I think I'm going to be using the same word that you drew on your chart, I'll call those concentration changes the deltas. Is that acceptable to you?
A. Yes.
Q. And you used these deltas to calculate the number of premature mortalities avoided in each county, correct?
A. Correct.
Q. And then you added up all these county numbers to get your total number, correct?
A. Correct.
Q. And in North Carolina, you calculated that in 2013 there will be 98 premature mortalities avoided if TVA were to emit at the so-called Clean Smokestacks equivalent case as compared to the 2013 base case, correct?
A. The number was 99, but...
Q. For $PM_{2.5}$ was 98, was it not?

A. $PM_{2.5}$ only?
Q. Right.
A. Yes, that was 98.
Q. And the 99 was ozone; is that correct?
A. That's correct.
Q. If you would turn in your book to TVA's Exhibit 377.
And Exhibit 377 is a deposition that was marked at your -- excuse me, is an exhibit that was marked at your deposition as well. And does it represent the $PM_{2.5}$ concentration levels in each county in North Carolina that were provided to you by Mr. Chinkin and Mr. Wheeler modeling the assumption that TVA would emit at the higher base case?
A. I believe that does. I'd have to look at the numbers specifically.
Q. And the longer document included a number of other states and this extract includes the North Carolina portion that begins on the second page with Alamance County, correct?
A. Yes. Correct.
Q. And then Exhibit 378 is similar. It is the modeling output that Mr. Chinkin and Mr. Wheeler provided you for each county in North Carolina showing the $PM_{2.5}$ levels in the county if TVA were to operate its plants at the lower controls case scenario, correct?
A. Correct.
Q. And they're all lower, aren't they?

A. Yes.
Q. And so the deltas would be obtained from subtracting the -- for each county the number in 378 from the number in 377, correct?
A. Correct.
Q. And Exhibit 371 is a table summarizing for each of the counties in North Carolina the results of that subtraction. Do you see that?
A. Yes, I do.
Q. And for example, in Alamance County, the model levels were 9.39 micrograms per cubic meter if TVA operated at the higher base case, dropping to 9.25 micrograms per cubic meter if TVA operated at the lower control case, correct?
A. Correct.
Q. For a difference of .14 micrograms per cubic meter, correct?
A. Correct.
Q. And this chart summarizes for each of those counties simply the results of the subtraction of Exhibit 378 numbers from 377 numbers, correct?
A. That's correct.
Q. And the highest delta in any county in North Carolina is .31 micrograms per cubic meter, isn't it?
A. I don't have all the numbers in front of me, but I'll accept that at this point.

Q. All right. The -- it's also in your book if you'd like to look at it, if you'd like to check it.
A. Okay. I'll presume your number is correct.
Q. The lowest number, the lowest, the smallest delta is projected to occur in Dare County which is on Page 1 and is 0.4 micrograms per cubic meter, correct?
A. 0.0.
Q. I'm sorry, thank you for correcting me.
A. 0.04.
Q. So the range of deltas you used in calculating 98 premature mortalities in North Carolina is 0.04 to 0.31 micrograms per cubic meter, correct?
A. Yes.
Q. Those are the amounts of the air quality improvements that your testimony is will avoid 98 premature mortalities, correct?
A. That's correct.
Q. And the baseline concentrations against which these deltas occur range from as low as about 5 micrograms per cubic meter in Dare County up to near 12 micrograms per cubic meter in Mecklenburg County, correct?
A. That's correct.
Q. In fact, Mecklenburg County was the highest level at 11.63 in the base case, correct?
A. That's correct.

Q. And in fact, this air dispersion modeling upon which you relied projects that 87 of North Carolina's 100 counties will have annual average $PM_{2.5}$ concentrations below 10 micrograms in the year 2013 even if TVA emits at the high levels projected by Dr. Staudt in his base case, correct?

A. That appears to be correct.

Q. And these deltas which are no greater than 0.31 micrograms per cubic meter, they are of a magnitude that's within the range of normal variation and monitoring instrument uncertainty, correct?

A. The reason why we conduct atmospheric modeling is to be able to determine these deltas between alternative control scenarios; and I think as Mr. Chinkin attested to, at times the modeling capability can be in advance of the monitoring capability.

Q. But in fact, these deltas which are no greater than 0.31 micrograms per cubic meter are a magnitude that is within the range of the normal variation and monitoring instrument uncertainty, correct?

MR. GOODSTEIN: Objection, Your Honor. Asked and answered.

THE COURT: I'll let him answer.

A. I'm not familiar with the different aspects of monitoring uncertainty. There's multiple types of instruments that measure $PM_{2.5}$ that have different degrees of measurement

error.
Q. If you could open the small notebook that I handed you, please, to Tab D. Is Tab D an article you wrote entitled, Using CALPUFF, C-A-L-P-U-F-F, to Evaluate the Impacts of Power Plant Emissions in Illinois Model Sensitivity and Implications?
A. Yes, it is.
Q. And your Harvard colleague, Dr. Spengler, was a coauthor of this article as well.
A. That's correct.
Q. If you would turn to Page 1073, please.
A. Okay.
Q. Did you write this in the left-hand column, the beginning of the last paragraph? Did you write: "An additional limitation is related to the difficulty of validating the model outputs. For our analysis, population weighted annual average concentration increments were on the order of 0.3 micrograms per cubic meter. Although impacts were as high as 0.6 micrograms per cubic meter close to the facilities and daily concentration variability at specific monitors might imply a larger effect on selected days, the magnitude is within the range of normal variation and monitoring instrument uncertainty."
A. I see that quote. I think the -- what this quote is trying to articulate is that when we apply atmospheric

dispersion models, there are different means of validating the outputs and that monitoring data, you know, cannot be used to directly validate these hypothetical scenarios and the outputs from these models, which is why we rely on other means of validation which I believe Messrs. Wheeler and Chinkin had attested to, the methods for validating the instruments -- I mean, the model.

Q. But it is true, sir, isn't it, that 0.3 to 0.6 micrograms per cubic meter is within the range of normal variation and monitoring instrument uncertainty?

A. At least for the instrumentation available circa 2002. Technology always does change.

Q. Thank you, sir. Now, the deltas that were -- are in North Carolina, they were primarily composed of sulfate, correct?

A. That's correct.

Q. And the nitrate component was a very small component, correct?

A. That's correct.

Q. And in fact, there are a number of counties in North Carolina where the modeling shows that when TVA reduced its nitrogen oxide emissions, nitrate levels actually went up; isn't that correct?

A. I'd have to look at the outputs, but that is likely the case because of the joint emissions of sulfur dioxide and

nitrogen oxide. It wouldn't be the case only from nitrogen oxide emissions in isolation. It's related to the complex secondary chemistry for sulfate and nitrate formations, but we certainly have documented that in other studies that SO_2 reductions can free up ambient ammonium to react with nitrates so you can reduce SO_2 emissions, reduce sulfate, but then increase nitrate by small amounts.

Q. But most of the mortality benefits that you calculated would occur in North Carolina are related to the specific particle sulfate rather than nitrate, correct?

A. That's correct.

Q. And sulfates are attributable to the specific pollutant sulfur dioxide, correct?

A. That's correct.

Q. I want to discuss the methodology you used to turn these $PM_{2.5}$ deltas into your calculation of premature mortalities avoided, but first I want to make sure I understand what you mean by the term premature mortality.

Is premature mortality a reduction in life expectancy as a result of an exposure?

A. That's correct. We know that the risk of death for people is 100 percent eventually. We're all going to die. So it's a matter of the degree of prematurity that would be exhibited due to an exposure.

Q. And when you talk about the degree of prematurity, you

mean the loss of somewhere on the order of one to three years of life expectancy, correct?

A. That's what the cohort studies have found on average would occur.

Q. Now, to calculate the number of premature mortalities avoided, you used what you call a concentration response function, correct?

A. Correct.

Q. And that's basically a formula, correct?

A. Correct.

Q. And the formula is that for every 1 microgram decrease in the annual average air concentration of $PM_{2.5}$, there will be a 1 percent decrease in mortality, correct?

A. Correct.

Q. And to arrive at your estimate of premature mortalities avoided, you simply multiply the population in each county by the baseline mortality rate for that county by the delta in concentrations by 1 percent to get the county mortality figure, correct?

A. That's correct.

Q. I want to do an example of this calculation based on Charlotte, North Carolina -- or actually, Mecklenburg County.

This is TVA Exhibit 384.

MR. GOODSTEIN: Your Honor, would it be possible to get a copy of this to put in front of the witness?

Q. Oh, it's in your book, sir, by the way, at 384 as well.
A. Okay. I've got it on the screen.
Q. We didn't mean to make your head spin there.
Now, from the documentation that you -- underlying documentation that you provided, it indicated that you used a population greater than 29 for Charlotte -- or excuse me -- my old home. I keep calling it that -- Mecklenburg County of 388,524, correct?
A. I don't remember all the individual numbers, but that looks correct.
Q. All right. And the baseline mortality rate of .0101?
A. Again, I'll take it at face value.
Q. And then the delta that was on the Exhibit 371, .17, correct?
A. Correct.
Q. And then the 1 percent translates just to a .01, correct?
A. Correct.
Q. And when you multiply those through, you come up with 6-2/3 premature mortalities in Mecklenburg County, correct?
A. Correct.
Q. If the overall $PM_{2.5}$ level is 11.63, your methodology indicates that during the year a little over 456 people in Charlotte will suffer premature mortalities from breathing $PM_{2.5}$, correct?
A. That's not a calculation that I would personally do, but

that -- if you do that calculation, that would be correct.
Q. Okay. So in other words, your calculation is that pollution -- excess pollution from TVA will cause 6-2/3 premature mortalities in Mecklenburg County and pollution from other sources in and out of North Carolina will cause another 450 premature mortalities, correct?
MR. GOODSTEIN: Objection, Your Honor. Mischaracterizes the testimony.
THE COURT: Let the witness answer it.
A. In the calculation you left out nonanthropogenic background and so this presumes that if you removed all sources, there would be 0 for $PM_{2.5}$ which is not correct. So you would have to subtract out nonanthropogenic background to get the attributable portion.
Q. Thank you for the correction. So what it means is that your calculation is that 6-2/3 people in Mecklenburg County will suffer premature mortalities from pollution alleged to be excessive and attributable to TVA while about 450 other people will suffer premature mortalities from the pollution that comes from all other places, correct?
A. Again, without accounting for background, that wouldn't be an appropriate calculation.
Q. And that means that throughout the year -- excuse me, throughout the state, when this calculation is replicated across all 100 counties, that means that thousands and

thousands of people are dying each year in North Carolina from breathing $PM_{2.5}$ at levels well below the standard; is that correct?

A. That is correct.

Q. And you understand that the methodology you used to calculate that TVA is responsible for 98 premature mortalities, you're aware that that methodology, if it's used on modeling output of Duke Energy and Progress Energy's North Carolina plants, it shows that they are causing on the order of 300 to 500 premature mortalities each year, correct?

A. I'm not sure if that's correct or not.

Q. You're not sure about that?

A. I'd have to look at the calculations.

Q. Do you recall being provided with that calculation at your deposition?

A. I don't recall offhand if those calculations were current emissions or future projected emissions, and what the other assumptions were in the calculations.

Q. I believe they were 2002 zero-out calculations.

A. Okay.

Q. And do you recall being provided with those calculations and being asked if they follow the methodology, same methodology that you used?

A. I do recall.

Q. And you are aware that when that methodology was

employed, it indicated 300 to 500 premature mortalities per year from the Duke and Progress plants?

A. Again, I don't recall the specific numbers, but I'll take it at face value.

Q. Thank you. Now, this health impact assessment you performed in this case is by no means the first one you've ever performed, is it?

A. That's correct.

Q. In 1999 you published a health impact assessment for Boston, correct?

A. Correct, for one power plant in Boston.

Q. And you and Dr. -- your colleague at Harvard, Dr. Spengler, both worked on that assessment, correct?

A. That's correct.

Q. And in that paper, you used a concentration response coefficient of 0.4 percent, correct?

A. I believe that's what we used for our base calculation, and we had some sensitivity calculations as well.

Q. In 2002 you and Dr. Spengler published a health impact assessment about Massachusetts, correct?

A. That's correct.

Q. And in that paper you used a mortality concentration response coefficient of approximately 0.5 percent, correct?

A. That's correct.

Q. Also in 2002 you and Dr. Spengler published a paper about

power plants near Washington, D.C., correct?
A. That's correct.
Q. And in that paper examining the health impacts of power plants around Washington, D.C., you used a coefficient of about 0.4 percent across the general population, didn't you?
A. Well, that I'd have to look at the paper. I mean, that specific paper was one in which we were looking at stratification across different levels of educational attainment, so that required using a different model and within our base calculation we used one function and we used a set of functions stratified by educational attainment.

I think it's also important to recognize that the scientific literature evolves over time and it would certainly not be prudent to develop -- to apply functions in 2008 that were developed in 1997.

So in each of these publications in the peer-reviewed literature, we re-examine the literature as a whole. In our 1999 paper, you know, which involved work a couple years prior initially. This was only shortly after the first couple of cohort studies had been published and prior to the Health Effects Institute's re-analyses of those studies. So there was less evidence available and less mechanistic evidence available about the health effects of $PM_{2.5}$.

New evidence arrived prior to our 2002 papers that led us to utilize different functions from the health effects since

re-analysis. There have been further publications that have come down the line.

So in each case we re-examine the evidence, consider the weight of the evidence and arrive at a separate function. So the fact that the functions have varied over time is simply an indication of the changing science and our utilization of that science.

Q. Well, I'm going to catch up to the present in a few minutes.

A. Okay.

Q. In the Washington, D.C. paper, you actually did two things. You both used conventional assumptions and then considered available evidence for differential effects on susceptible subpopulations, correct?

A. That's correct.

Q. And you used three different coefficients for the -- what you called the susceptible subpopulation, .81 percent with people less than a high school education, .44 percent for people with a high school education, and essentially zero for people with greater than high school education, correct?

A. Actually, my numbers are slightly different, but it was --

Q. Well, what are they?

A. 8.5 percent for less than high school education, 4.5 with high school education, and not statistically significant but

.3 percent for those with more than high school education.
Q. And those were all for 10 microgram changes, correct?
A. That's correct.
Q. To make them apples to apples with the numbers you're using here, you would call them .8, .4 and 0, correct?
A. Correct.
Q. And these different coefficients for subpopulations translated to an overall coefficient of about .4 percent across the general population, correct?
A. That's correct. Again, it's so we could use a value that was commensurate with the stratified calculations so we had an apples to apples calculation in the paper.
Q. Still in 2002, you and Dr. Spengler and others published a paper about Illinois, correct?
A. That's correct.
Q. And the purpose of that Illinois paper was to determine the influence of key atmospheric modeling assumptions on health based conclusions, correct?
A. That's correct.
Q. And as part of your analysis in that paper, you applied a concentration response function for premature mortality, correct?
A. That's correct.
Q. And the concentration response function for premature mortality that you and Dr. Spengler used in the 2002 paper

about Illinois was 0.5 percent, correct?
A. I believe that's correct. I'm looking for the dimension here.
Yeah, that paper was largely focused on atmospheric modeling. This was a side calculation in the discussion section.
Q. Using 0.5 percent?
A. I'm looking for -- I presume that's correct.
Q. I believe it might be on Page 1069.
A. Thank you, I found that.
Q. I'm sorry, I missed your answer. Did you confirm that you used 0.5 percent?
A. Yes, I did.
Q. I'm sorry, I didn't hear you.
In 2003 you and Dr. Spengler and others published a paper about Georgia, correct?
A. That's correct.
Q. And that Georgia paper examined emissions from seven power plants located near Atlanta, Georgia.
A. That's correct.
Q. And as part of your Georgia analysis, you did an illustrative evaluation of the approximate magnitude of health impacts associated with these seven Georgia power plants using a coefficient of 0.6 percent, correct?
A. That's correct.

Q. In 2005 you and Dr. Spengler prepared a report evaluating public health implications relating to a Wisconsin power plant, correct?
A. That's correct.
Q. And in the Wisconsin power plant analysis, you used a coefficient of 0.5 percent, correct?
A. That is correct.
Q. In 2007, a study -- in May 2007 a study was published that you authored entitled Quantifying the Efficiency and Equity Implications of Power Plant Air Pollution Control Strategies in the United States, correct?
A. Correct.
Q. And you submitted that article for publication in September 2006?
A. That's correct. The analyses, however, were conducted in the summer of 2005 and in the winter of that year.
Q. And it was accepted for publication in January of 2007.
A. That's correct.
Q. And actually published in May 2007.
A. That's correct.
Q. And in this 2007 article, you used a concentration response function coefficient of 0.6 percent, correct?
A. That's correct. Since this involved fairly complex simulation modeling -- you know, as the new scientific evidence in 2005 and 2006 unfolded, it was computationally

infeasible to rerun the models, so the outputs that we generated were based on that work in the summer of 2005 and really completed in that winter.

Q. To summarize the concentration response function and coefficients you've used in your past work, the 1999 Boston study was .4 percent; the 2002 Massachusetts study was .5 percent; the 2002 Washington, D.C. study was .4 percent; the 2002 Illinois study was .5 percent; 2003 Georgia study was .6 percent; 2005 Wisconsin study was .5 percent; and the 2007 national study was .6 percent, correct?

A. This is commensurate with the range of central estimates that have been reported within the American Cancer Society study which the authors report generally between .4 and .6 percent.

Q. And before this case, you had never conducted a risk analysis calculating premature mortalities associated with $PM_{2.5}$ exposure using a coefficient higher than .6 percent, correct?

A. Correct.

Q. And the central tendency of the coefficients you have used in your past studies is about .5 percent, correct?

A. That's correct.

Q. And in your opinion, a concentration response coefficient of .5 percent for a $PM_{2.5}$ premature mortality calculation is not an unreasonable or implausible one to use, is it?

A. It's within the range of plausible values, I think. As many at EPA and elsewhere had done, we had relied to date on the American Cancer Society's study. I think EPA and others recognize that this was a much larger study than the Harvard Six Cities study and that provided it with many advantages.

There was growing recognition starting with the publication by Jared in 2005 that the method for exposure assignment in the American Cancer Society study may have contributed to some exposure misclassification, basically using one monitor to represent a fairly large geographic area; and in these kind of studies, that type of misclassification tends to bias your estimates downward.

I think there was also a growing recognition both from the stratified analyses on education, a nice paper by O'Neil and colleagues in 2003, that the socioeconomic status effect was real and important.

And between those two effects, I think there was growing recognition in the scientific community, as well as within myself and Dr. Spengler's opinions that the values from the American Cancer Society study, the .4 to .6 values were likely systematically underestimated. And I think that is reflected in the expert elicitation outputs in the value that we used in this report; and in fact, in -- I believe I've got four or five papers that are submitted for publication now going forward that are using this 1 percent or in some cases higher

function.

So I think the scientific understanding about these effects has evolved over time. I think there was actually an inflection point in '05 and '06 when people started to recognize some of the downward biases of the American Cancer Society study.

Q. So in health impact assessments published from 1999 through 2007, you always used a coefficient on average of about .5 percent, correct?

A. Yes -- or I would call it analyses prepared between 1997 and 2005.

Q. But here you used 1 percent, correct?

A. Right.

Q. And using 1 percent instead of .5 percent doubles the number of premature mortalities you calculated, correct?

A. That's correct.

Q. And the primary basis of this choice of a 1 percent coefficient was that you derived it from a so-called expert elicitation study funded by EPA, correct?

A. It was that 1 percent was corroborated by the expert elicitation study; but also, as I indicated, by our own reading of the literature, by the growing understanding about the effects of exposure misclassification within the ACS study, as well as the educational attainment effect.

Q. If you'd turn to your deposition transcript, please.

It's Exhibit Number 368.

A. My colleague, Mr. Woodall, asked you at your deposition, "You used a 1 percent function" -- this is -- I'm sorry, I'm on page 44.

Page 44, line 6, you were asked: "You used a 1 percent function in this report. Why did you use a different function in this report than the one that was used in the earlier reports?"

And to begin your answer, you said, "The primary basis was an expert elicitation study that came out in the fall of 2006." Correct?

A. That is what I stated there. Subsequent to the deposition I did go back and look through sort of the sequence of report preparation and realized that these things actually happened in parallel. We had certainly begun determining the appropriate concentration response function and had come up with our 1 percent value prior to the publication of that study in the fall of 2006. We were obviously aware that the study had been ongoing for a period of time and had heard of some of the results and so I don't remember all the time sequences involved. You know, that was clearly an important corroboratory basis and something that we looked at carefully.

I think as I also said on Page 45 of the deposition is that this also led us to reread the literature and recalibrate our thoughts on that literature.

Q. And after you gave your deposition, you read it, didn't you?
A. My deposition?
Q. You read the transcript, didn't you?
A. Yes, I did.
Q. And you made a number of changes to it, didn't you?
A. Yes, I did.
Q. And then you signed it, didn't you?
A. Yes, I did.
Q. And you didn't change that testimony, did you?
A. I suspect I did not.
Q. Would you like to review your changes to find out?
A. No, that's all right.
Q. Thank you. Now, the way you derived your 1 percent coefficient from the so-called expert elicitation was to take a simple average of the medians of each of the 12 estimates given in that study, correct?
A. That's correct.
Q. I'd like you to look at TVA Exhibit 374.
What is Exhibit 374?
A. That is a cover page that was appended on to the expert elicitation report produced by Industrial Economics that I referred to earlier.
Q. And does it not say -- is this put out by EPA itself?
A. Yes, it was.

Q. And does it not say, "This expert elicitation report was prepared in support of the characterization of uncertainty in EPA's benefits analyses associated with reductions in exposure to particulate matter pollution. As recommended by the National Academy of Sciences, EPA is using expert judgment as part of an effort to better describe the uncertainties inherent in any benefits analysis. This report and its findings are intended solely for this purpose."

Is that what it says?

A. Yes. There's also, if you read in the executive summary of that same report, this is on Page 4 of 109. The report says, Expert elicitation uses carefully structured interviews to elicit from each expert his best estimate of the true value for an outcome or variable interest as well as his uncertainty about that true value," and goes forward from there.

I can't speculate on EPA's intent on the cover page other than that, as the experts all indicated, health effects down to 7 micrograms per cubic meter. This, you know, certainly would be problematic for the NAAQS setting process. EPA has used the expert judgment outputs, including their central estimates, in their regulatory impact analyses conducted since that time looking at new source performance standards for -- I'm blanking on the source sector, but it was their regulatory impact analysis done in March of this year as well as in -- some of their other regulatory impact analyses of the last

couple years.

So EPA has used these values directly, including the central values for regulatory impact analysis and my -- I can't read their mind, but I expect that this was overlaid on their report related to concerns about the NAAQS.

Q. Although we can't read the EPA's mind, we can read EPA's words and EPA's words were that this so-called expert elicitation was intended to better describe the uncertainties inherent in any benefit analysis, and the report and its findings are intended solely for that purpose, correct?

A. Yes. And the quotation that I read was also from that EPA report.

Q. And in the body of the report itself, it states that "the purpose of this project is to provide a more complete characterization, both qualitative and quantitative, of the uncertainties associated with the relationship between reductions in ambient $PM_{2.5}$ and mortality," correct?

A. That's correct. And you know, part of characterizing the uncertainty distribution for each expert involves certainly their upper and lower bound values, their 5th and 95th percentiles, but a key component of that is also the center of that distribution. So it's meant to capture the degree of uncertainty within the expert arena about a lot of these questions as well as areas of consensus and lack of uncertainty.

Q. There is a methodology for combining a number of studies together called a meta-analysis, correct? M-e-t-a-analysis.
A. That's correct.
Q. A meta-analysis is a systematic and quantitative synthesis of the epdemiological evidence from a number of published studies, correct?
A. That's correct.
Q. I want to read you a few sentences from the Reference Manual on Scientific Evidence put out by the Federal Judicial Center, the chapter on epidemiology, and ask if you agree with this.

"Meta-analysis is a method of pooling study results to arrive at a single figure to represent the totality of the studies reviewed. It is a way of systematizing the time-honored approach of reviewing literature which is characteristic of science and placing it in a standardized framework with quantitative methods for estimating risk. In a meta-analysis, studies are given different weights in proportion to the sizes of their study populations and other characteristics."

Is that an accurate description of what a meta-analysis is?
A. Yes, it is.
Q. To select the concentration response function coefficient of 1 percent that you used here, you did not conduct a

meta-analysis, did you?
A. Well, there's a couple levels of response here. I mean, one is we conducted that precise form of meta-analysis for all other health outcomes and all other -- I mean, for ozone, for all outcomes and for the morbidity outcomes for PM.
Q. And just to be clear, I was not asking about the other ones. I was asking about the $PM_{2.5}$ coefficient of 1 percent. Did you conduct a meta-analysis to arrive at that figure?
A. But the point is that these meta-analytic techniques are meant when one can pool the evidence across independent samples with a large sample size that can then be used to synthesize the evidence. So for, you know, for example, cardiovascular hospital admissions, there were, I believe, 51 studies so that was amenable to this sort of assessment. For PM mortality there were many publications that stemmed from two key cohorts. These were not independent observations and a meta-analysis text would say that it is inappropriate to just simply statistically pool independent observations.
Moreover, if one considers the expert elicitation distributions as, you know, potential key inputs to this process, the expert elicitation report itself explicitly stated that statistically combining expert opinions as opposed to independent publications is not well justified by, you know, common practice within expert elicitation. And that's why EPA, when it's been utilizing expert elicitation outputs

in its regulatory impact analyses, has not formally done pooling meta-analyses of those studies.

And so we felt that given the nature of the evidence, given the nature of the publications and the expert elicitation, that simply throwing them into an assessment without a more rigorous consideration of what was entailed in each each of those estimates would not have been appropriate.

Q. I may have stayed up too late reading epdemiological studies and I'm not phrasing my questions well, I suppose. I meant to ask you, sir, did you or did you not conduct a meta-analysis to arrive at the figure of 1 percent to use as the concentration response function coefficient for your $PM_{2.5}$ premature mortality calculation?

A. I mentioned earlier that there were two levels of my response. The second level is that meta-analysis takes many forms and statistically pooling studies through inverse variance weighting is one form of meta-analysis. Another involves reading and synthesizing the evidence and coming to a determination of the pooled understanding without such a formal approach. EPA in its regulatory impact analysis describes its approach as meta-analytic, but does not do this form of statistical pooling across the PM cohort studies for similar reasons.

So, you know, similar to how there are different levels of uncertainty analysis I described earlier, there are

different forms of meta-analysis. So I would consider what we did as a meta-analysis even if not an inverse variance weighted meta-analysis.

Q. You agreed earlier that a meta-analysis is a systematic and quantitative synthesis of the epdemiological evidence from a number of published studies, didn't you?

A. Yes, I did.

Q. You did not conduct a systematic and quantitative synthesis of the epdemiological evidence from a number of published studies in order to arrive at the 1 percent figure, did you?

A. We did a systematic review of the literature and we did a quantitative assessment. So I would say that, yes, we did do that form of meta-analysis as described. We did not do inverse variance weighting or some of the other techniques that I've applied in previous publications just given the nature of the evidence, the number of studies and the methods that were appropriate under the circumstances.

Q. And you agreed with me a moment ago that in a meta-analysis, studies are given different weights in proportion to the sizes of their study populations and other characteristics. Did you do an analysis in which you gave different weights to the different studies in proportion to the sizes of their study population and other characteristics?

A. Well, this was in fact the reason why in our earlier

publications we had given very large weight to the American Cancer Society study outputs because it's a much larger cohort than the Harvard Six Cities study. We also needed to take into consideration some of these potential biases and issues that do not lend themselves to, again, simply doing these quantitative weights and ignoring other attributes of the studies.

So, you know, the formalized weighting approach inherent in inverse variance weighted meta-analysis we did not do for this one endpoint on one pollutant, but it was not justified under the circumstances.

Q. Would it be fair to say you did a meta-analysis light?

A. I'd say it would be fair to say that I did what is appropriate given the scientific evidence available.

Q. But you did not do a formal meta-analysis that involved any sort of statistical analysis, did you?

A. I think, as many others have concluded, that sort of a blind pooling of studies in this context is inappropriate, and that's why I'm not aware of any publications of regulatory impact analysis where for PM cohort mortality such a pooling has been done. I think it's not -- not a well justified approach.

I would add parenthetically that had we done that calculation and selected, you know, a representative study from the American Cancer Society cohort and a representative

study from the Six Cities cohort, we would have arrived at a value greater than 1 percent. But we didn't feel that that was the most appropriate means for doing this calculation and representing the totality of scientific evidence.

Q. You think you would have arrived at a value greater than 1 percent if you had done the calculation you didn't do?

A. Yes, I would have. I think one can -- I mean, I've done these sorts of meta-analyses many times over the last 12 years. I think if you just look at the confidence intervals described within the different cohort studies, they are roughly of comparable magnitude. So, you know, I could do the calculation if I had a spreadsheet in front of me, but eyeballing the confidence intervals and looking at the central estimates, I think it's pretty clear that one would arrive at a value higher than 1 percent.

Q. No where in your report did you describe the methodology by which you came up with the 1 percent figure as a meta-analysis, did you?

A. I'm not aware of the language that I used, but I'd say, you know, within health impact assessments, constructing concentration response functions involves meta-analyses. It's an inherent part of the process.

Q. EPA periodically reexamines the National Ambient Air Quality Standards, correct?

A. That's correct.

Q. And in 2006, EPA completed its review of the $PM_{2.5}$ National Ambient Air Quality Standard, correct?
A. Correct.
Q. And for the annual standard, EPA decided to keep it where it already was, at 15 micrograms per cubic meter, correct?
A. Correct.
Q. And that was where it had been set back in 1997, correct?
A. That's correct.
Q. And in 1997, EPA was provided with a quantitative risk assessment, the kind of risk assessment you have performed here, as part of a standard setting process, correct?
A. That's correct.
Q. But in 1997, EPA considered it to be too limited to serve as a quantitative basis for decisions on the standard level, correct?
A. That's correct. EPA uses those tools in regulatory impact analyses, but it's not a primary basis for NAAQS.
Q. Regular impact analyses occur after the standard has already been set, correct.
A. Well, that occurs in separate and in other settings. For the Clean Air Interstate Rule, for example, that occurred -- that analysis was prior to the setting of the $PM_{2.5}$ standard, but in parallel to the needs to reduce $PM_{2.5}$ concentrations.
Q. And in this most recent round of setting the 2.5 ambient air quality standard, another quantitative risk assessment,

the kind of risk assessment you performed here, was presented to EPA, correct?

A. Correct.

Q. And the EPA administrator determined that this quantitative risk assessment did not provide a reliable basis to determine what specific quantitative revisions to the standards would be appropriate, correct?

A. That's correct.

Q. What the specific words that the EPA administrator used were that the risk assessment did not provide a reliable basis, correct?

A. I'll take that at face value there.

Q. I want to ask you some questions now about uncertainties. Any health impact assessment of the sort you performed in this case will contain uncertainties, correct?

A. That's correct.

Q. Although you chose 1 percent as your coefficient here, there is uncertainty associated with that coefficient, isn't there?

A. That's correct. And that was described in our report.

Q. There are clearly numerous plausible estimates of mortality concentration response functions based on reported confidence intervals, alternative statistical models within studies, use of different studies, and alternative assumptions about particle constituent toxicity, correct?

A. That's correct.
Q. There is also uncertainty about the shape of the concentration response function, whether it is linear or non-linear, correct?
A. That's correct.
Q. A threshold -- you discussed earlier issues about a threshold. A threshold is a concentration level below which it is expected that effects are not observed, correct?
A. That's correct.
Q. In your opinion -- excuse me. It's your opinion that the available evidence does not support or refute the existence of a threshold for chronic effects for $PM_{2.5}$, correct?
A. That's correct.
Q. Thus from the available evidence, one cannot rule out the possibility that there is a threshold exposure level for $PM_{2.5}$ below which exposure does not cause serious health effects such as premature mortality, correct?
A. Correct. One cannot rule out the existence of that threshold at levels that we have never observed within the cohort studies. At levels at which we have empirical evidence, I think we can quite clearly state that no threshold does exist.
So as mentioned previously, studies like the Six Cities follow-up in 2008 demonstrated effects down to 7 micrograms per cubic meter. Whether there are effects down below 7,

there is no empirical evidence to support or refute a threshold. I think as I described it, it does seem implausible that that lowest level that had been observed in the Six Cities study would coincidentally be precisely the value at which this curve which was a straight line from 30 or 40 down to 7 would abruptly and immediately change.

So I would think the best available scientific information would indicate that the curve continues on in its form as observed and would be the most reasonable assumption. Although, given a lack of empirical data below 7, I think one could not rule out non-linearities.

Q. If you could turn back to Exhibit 368 in your book which is your deposition transcript.

A. Uh-huh.

Q. And it will be Page 73.

Were you asked, sir, "Question: It's your opinion that the available evidence does not support or refute the existence of a threshold for chronic effects for $PM_{2.5}$; is that correct?"

And you answered, "That's correct," didn't you?

A. That's correct.

Q. Now, as I understand it, epdemiological evidence forms the centerpiece of the concentration response function which you used to calculate premature mortalities which you contend result from exposure to $PM_{2.5}$, correct?

A. That's correct.
Q. Epidemiology is a science that is used to examine the pattern of disease in human populations, correct?
A. That's correct.
Q. Epidemiologic studies evaluate the relationship between health outcomes and ambient concentrations as recorded by central site monitors, correct?
A. Not all epidemiologic studies are structured that way, but many of the ones on which we relied are structured that way.
Q. And an epidemiologic study may show an association between exposure to an agent and some health outcome like, for instance, an association between higher $PM_{2.5}$ concentrations in the area and higher mortality rates, correct?
A. That's correct.
Q. But an association -- but an association is not equivalent to causation, is it?
A. It is not. And I would -- I would say one single epidemiologic study in isolation without any other evidence should not be viewed as evidence for causation; but a large body of epidemiologic studies consistent over time, locations and methods with other corroborating evidence can then be viewed as evidence for causation.
Q. But an association itself is not equivalent to causation, is it?

A. That's correct.
Q. And an association identified in an epidemiologic study may or may not be causal, correct?
A. Correct.
Q. To arrive at a conclusion of causation, you need to rely on both epidemiological evidence and toxicological evidence, correct?
A. That's correct.
Q. And you do not hold yourself out to be an expert in toxicology, do you?
A. No, I don't.
Q. Nor do you have any training in medicine or biology in connection with assessing the biological mechanisms of health effects, do you?
A. No, I do not.
Q. And you have acknowledged in your public writings that the assumption that mortality associations shown in the epdemiological studies reflect a causal relationship for $PM_{2.5}$ is a substantial uncertainty, correct?
A. That's correct. I would say that I suspect that was from one of my either 1999 or 2002 papers. I would say that as the scientific evidence has grown substantially in the intervening years, my opinion on that question would change.
And I think, you know, if you look at our 1999 paper, we consider the time series evidence as our primary central

estimate because there had not been sufficient corroboratory studies of the cohort evidence. As the cohort evidence has grown and the biological plausibility evidence has grown, I think I have gotten much better confidence in the causal association as well as our understanding of the magnitude of the relationship.

I think that's also reflected in the expert elicitation study where the experts were asked the probability of causality given the totality of epidemiologic and toxicologic evidence, and I believe 10 of the 12 experts expressed causal probabilities over 90 percent, some on the order of 95 to 99 percent. You'll never get an academic to say that anything is 100 percent certain. But, you know, there's strong belief within the scientific community that this is a very well documented causal relationship.

Q. In October 2006, the EPA administrator determined that he was not prepared to make the assumption that associations between mortality and $PM_{2.5}$ are causally related at levels as low as the projected 2013 North Carolina ambient concentrations at issue in this lawsuit, correct?

A. That's correct.

Q. Fine particulate matter, $PM_{2.5}$, is composed of many different kinds of substances, correct?

A. Correct.

Q. Sulfate and nitrate are two of the components of $PM_{2.5}$,

but there are many others as well, correct?
A. That's correct.
Q. And the different components of PM2.5 are not all equally toxic, are they?
A. It seems unlikely they would all be equally toxic.
Q. And we do not have definitive information about the relative toxicity of the different particle constituents of $PM_{2.5}$, do we?
A. No, we do not. No constituents have been either exonerated or specifically implicated.
Q. Thus there remains uncertainty about the quantitative relative toxicity of the key constituents of $PM_{2.5}$, correct?
A. That's correct.
Q. In addition to uncertainties in your health impact analysis, there are uncertainties in what I call information upstream of your analysis. For example, as we discussed a few minutes ago, you were relying on Dr. Staudt's emissions estimates, correct?
A. Correct.
Q. And if Dr. Staudt's projections of the amount of TVA's expected 2013 sulfur dioxide emissions are in error by, say, 100,000 or 200,000 tons, that would have an effect on your analysis, wouldn't it?
A. It would. It would certainly influence the temporal distribution of the benefits. If controls were put in place

earlier than when we had modeled, those benefits would start accruing prior to 2013 and the magnitude of benefits, you know, additional to that after 2013 would be smaller.

MR. LANCASTER: May I approach the map, Your Honor?

THE COURT: Yes.

Q. You're here. If the evidence shows, sir, that Dr. Staudt's emissions projections are substantially overstated, particularly overstated at TVA's Johnsonville, Bull Run and Kingston plants in eastern North Carolina, that would have an effect on the calculations you made, wouldn't it?

A. Yes, it would.

Q. Thank you, sir.

In your view, the two epdemiological studies that give the primary evidence for the mortality effects are the American Cancer Society study and the Harvard Six Cities study, correct?

A. That's correct. For $PM_{2.5}$.

Q. Thank you. The Harvard Six Cities study, as its name suggests, involved six communities, correct?

A. Correct.

Q. What were they?

A. Oh, I'll have to recall. Portage, Wisconsin; Kingston/Harriman, Tennessee; Watertown, Massachusetts -- which were the others?

Q. Watertown, Massachusetts; Portage, Wisconsin; Steubenville, Ohio --
A. Steubenville.
Q. -- Topeka, Kansas; Harriman, Tennessee; and South St. Louis, correct?
A. Yes, that's correct.
Q. I happen to live near Harriman, Tennessee, and I cannot see why it is described as a, quote, unquote, city, but it is included in the Harvard Six Cities study, correct?
A. Right. Portage, Wisconsin, is not a terribly large city either, but every one is included.
Q. The primary limitations of the Harvard Six Cities study were the small number of subjects from a small number of study areas, correct?
A. Correct.
Q. And the six communities in the Harvard Six Cities study clearly cannot represent all the distributions of all the populations in the United States, can they?
A. No, but I think it has the advantage of having a very systematic selection process for cohort members that were meant to be representative of those locales and those locales were selected to be representative of broader geographic areas. So, you know, in comparison to the ACS study, there was a very explicit and careful attempt to get populations that were meant to be more representative of the United

States.
Q. But the concern about the -- pardon me. The concern about the Six Cities study is its lack of representativeness, correct?
A. Yes.
Q. Now, the American Cancer Society study, it actually showed that there's only a small and insignificant association between $PM_{2.5}$ exposure and mortality for people with more than a high school education, correct?
A. That's correct.
Q. So is that telling us that the best thing to do to protect ourselves from air pollution is stay in school?
A. I think what it says, along with many other studies looking at other risk factors, smoking, anything across the board, is that socioeconomic status matters and other vulnerability attributes matter. Clearly walking across the stage and getting your high school diploma doesn't immediately confer you with magical protection from air pollution. But what this is is an indication of those of lower socioeconomic status, for a variety of susceptibility and possibly exposure related reasons, would be at greater risk from air pollution.
Q. And I wouldn't have thought that my college degree was going to protect me from air pollution, but is the Harvard Six Cities study showing that all of the people in the study who have college degrees or who have greater than high school

education, that they simply are not statistically associated, there's no rise in death rates for rises in pollution levels of $PM_{2.5}$?

A. I believe that's correct as well. I think both the Six Cities and the ACS studies show that socioeconomic gradient with highest effects in those with the lowest level of education.

Q. Neither the ACS study nor the Harvard Six Cities study show a significant positive association between ozone and mortality, correct?

A. Publications to date indicate that, that's correct.

Q. In other words, neither the ASC study nor the Harvard Six Cities study supports the argument that ozone causes mortality, do they?

A. They do not contain evidence that would show that long-term exposure to ozone contributes to mortality increments.

The time series literature, which is quite large, shows that ozone exposure, daily excursions of ozone exposure are associated with increases in mortality.

And, you know, one of the issues certainly present in the Harvard Six Cities study is that there was a relatively small gradient of ozone exposure across the different communities. I think as the modelers previously indicated, ozone can travel a fairly large distance. And, you know, if one lacks an

exposure gradient in a cohort study, there's no ability to discern effects even if those effects were to exist.

I think the American Cancer Society study has a bit more heterogeneity. Some of the more recent findings from the extended following are showing significant effects on ozone long-term mortality. But, you know, that evidence is not really, you know, as substantially out there in the world yet. We relied solely on the time series literature.

Q. You relied on the ACS study and the Harvard Six Cities Study to support your opinion that mortality is caused by $PM_{2.5}$ exposure, correct?

A. That's correct.

Q. But then you relied on different studies to support your opinion that ozone exposure causes mortality, correct?

A. Time series and cohort studies contain potential overlapping effects. The effects in the cohort studies are generally much larger because it can include the effects of long-term exposure. So for PM there's very strong evidence from time series studies of mortality effects, but we were worried about double counting so we left out the time series effects for PM and solely relied on the cohort evidence.

So, you know, it's, because of the degree of overlap, inappropriate to include both and so that's why with ozone we only relied on the time series estimates. With PM there's both time series and cohort estimates and we solely relied on

the cohort so we didn't overstate the effects.
Q. So you relied on different studies for the ozone mortality, correct?
A. That's correct.
Q. And the studies that you relied on for your $PM_{2.5}$ mortality calculations, they don't support a causal -- any sort of causal connection between ozone and mortality, correct?
A. Well, that's an overstatement. I think they don't have the evidence available to discern such an effect, and air pollution can cause effects both due to acute changes in exposure and due to chronic exposure and it's not an either/or proposition. So even if there were not evidence of long-term effects of ozone exposure on mortality, there can still be evidence of short-term effects of exposure on mortality.
Q. You're familiar with what is known as the Lipfert, L-i-p-f-e-r-t, Veterans Administration study, correct?
A. Correct.
Q. It was a cohort study of 50,000 U.S. veterans, correct?
A. Correct.
Q. And the Lipfert VA study showed no significant association between particulate matter and mortality for any of the various measures used for particulate matter, correct?
A. That's not correct. It depends on the publication. As indicated in that table by Pope and Dockery, one of the

Lipfert publications showed an effect between PM and mortality on the order of 1.5 percent increase in deaths per microgram per cubic meter increase, but also contained a large number of sensitivity calculations. So -- but in their -- certainly in their first view, which was a preliminary look at the cohort, no effect was observed. In their follow-up analysis, an effect was observed, but in other statistical models that effect was blunted.

Q. You're familiar with the Enstrom, E-n-s-t-r-o-m, study of 50,000 older Californians, aren't you?

A. Yes.

Q. For the nearly 20 year period from 1983 to 2002, the Enstrom study showed no association between particulate matter exposure and mortality, correct?

A. That's correct. I think I and many others had methodological concerns about the Enstrom study and I think that is reflected in some peer-reviewed publications reflected in the California Air Resources Board's summary of PM concentration response function evidence as well as EPA's evaluation of this literature.

Q. But to be clear, I was correct in stating that the Enstrom study showed no association between particulate matter exposure and mortality, correct?

A. That's correct. Again, with some large methodological concerns.

Q. The ACS study, American Cancer Society, in the extended version of that study, the long-term average $PM_{2.5}$ concentrations across the cities in the studies was about 17.7 micrograms per cubic meter, correct?
A. I believe that was the average across all cities and all dates.
Q. In the Harvard Six Cities study, the long-term average $PM_{2.5}$ concentration across the cities in the studies is about 18 micrograms per cubic meter, correct?
A. I think it depends on which follow-on period you're looking at. I think in at least one of the earlier Six Cities publications, that was the average across cities and dates.
Q. And in terms of $PM_{2.5}$ levels, you acknowledge, do you not, that more uncertainty exists at the lower end of the $PM_{2.5}$ concentration ranges, correct?
A. That's certainly the case, I think. You know, the fact that the average values were 17 or 18 obviously implies that roughly half of the population were exposed to levels below that and effects were exhibited below those levels. So I think we need to look at the range of concentrations in the studies to formulate our opinions about the functions and thresholds, not simply at the average.
Q. But more uncertainty exists at the lower end of the concentration ranges, correct?
A. That's correct.

Q. You're familiar with the National Ambient Air Quality Standard for $PM_{2.5}$ that we discussed a few minutes ago, correct?
A. Yes.
Q. Federal law requires EPA to set a National Ambient Air Quality Standard at a level which in the judgment of the EPA administrator is requisite to protect the public health with an adequate margin of safety, correct?
A. That's correct.
Q. In October 2007, EPA finalized its review of the $PM_{2.5}$ National Ambient Air Quality Standards, correct?
A. Correct.
Q. And EPA retained the standard at 15 micrograms per cubic meter, correct?
A. That's correct.
Q. And it did so after reviewing what you referred to as the voluminous 2000-page long criteria document, correct?
A. That's correct. Although CASAC, the Clean Air Science Advisory Committee, as well as EPA staff had recommended that given the scientific evidence, the standard be lower. The administrator chose not to follow that guidance.
Q. I had a feeling you'd bring up CASAC. CASAC's recommendation was that the standard should be set at 13 or 14 instead of 15, correct?
A. That's correct.

Q. And CASAC informed EPA, "The uncertainties would increase rapidly below an annual level of 13 micrograms per cubic meter," correct?
A. That was their statement, yes.
Q. And all of the counties in North Carolina where you predict premature mortalities to occur because of exposures to $PM_{2.5}$, all hundred counties are projected to have $PM_{2.5}$ levels below 13 micrograms per cubic meter in 2013, correct?
A. That's correct.
Q. In fact, they're all projected to be below 12, aren't they?
A. That's correct.
Q. In fact, 87 of the 100 counties are projected to be below 10, correct?
A. That's correct.
Q. So there are a number of sources of uncertainty surrounding your premature mortality opinion in this case, correct?
A. Yes. As acknowledged, there are clear uncertainties and our estimates represent the best estimates given the available science.
Q. And you consider that in a peer-reviewed journal publication, it would be very important to give quantitative uncertainty bounds or sensitivity calculations, correct?
A. That's correct.

Q. And here, to do a formal quantitative assessment of uncertainties would require propagation of uncertainties through all the stages of the analysis, correct?

A. Yes. It depends also, in part, on what level of uncertainty analysis one wishes to conduct. And I think I alluded to this morning that WHO described different levels of uncertainty analysis for what they would call a Tier III uncertainty analysis which is formal quantitative propagation using Monte Carlo analysis. The only appropriate way to do it would be to consider uncertainties in each part of the assessment and then to formally combine them rather than in single components of the assessment.

I think for other levels of uncertainty analysis, I think for other of the tiers that the WHO had laid out, different forms of quantitative or qualitative uncertainty analysis are justified.

Q. If you would turn back to your deposition which is Exhibit 368. And I'm on Page 112.

Are you with me yet, Dr. Levy?

A. Yes, I am.

Q. On line 8 you were asked -- excuse me, on line 5 you were asked, "Why didn't you include an opinion about the extent of the uncertainty of your numbers in your report?"

And your answer was, "We were asked to give an opinion on the most credible estimate of the benefits of emission control

so that was the opinion that we had offered.

"Question: Do you consider it important in evaluating that estimate to present the uncertainty that is associated with the estimate?

"Answer: I would consider it in a peer-reviewed journal publication very important to give quantitative uncertainty bounds or sensitivity calculations which, as you can see, we offered up in our previous assessments. In this case that was not a question that was asked and we presented a qualitative description of the fact that estimates could be larger or smaller since to do a formal quantitative assessment would require propagations of uncertainty through all the stages of the analysis which was beyond the scope of what was within our assessment."

Was that your testimony, sir?

A. That was.

Q. And here you have not done a quantitative uncertainty bounds or sensitivity calculations that would be very important to do in a peer-reviewed journal publication, correct?

A. Well, in a peer-reviewed journal publication, part of what we're seeking to do in propagating and then essentially segmenting out uncertainty is to make recommendations for future research directions, and that is something that has been emphasized in a number of our previous publications.

This is a very different context in which we were asked to render our best judgment of what the impacts would be and the benefits would be of reducing the excess emissions from the TVA facilities. You know, we did describe for PM mortalities the uncertainties and gave the explicit quantitative implications within a boundary calculation to demonstrate some of the possible uncertainties.

And I think as, you know, the WHO has emphasized and multiple National Resource Council committees have emphasized, you know, uncertainty analysis at the level that is done within the peer-reviewed literature is not always called for in a decision context. That just because computers can very readily crank out these complicated uncertainty propagations doesn't mean that helps people make decisions; and that the degree of uncertainty characterization, the level of it and the nature of the information depends on the context of the decision. And I think that was articulated first probably in the 1994 National Research Council report in the Blue Book and then described by the WHO and many others.

Q. Sir, in this particular case regarding the opinions that you've given in this lawsuit, you did not give quantitative uncertainty bounds or sensitivity calculations of the type that you would consider to be very important to give in a peer-reviewed journal publication; is that correct?

A. That is correct.

MR. LANCASTER: Your Honor, I'd like to move into evidence Defendant's Exhibits 371, 377, 378, 384, and 374. Sorry about getting out of order there.

MR. GOODSTEIN: Your Honor, these calculations apparently have been put together by counsel for TVA and I'm not sure there's an adequate foundation for these calculations that have been put together by counsel. Obviously not a qualified expert in this case.

MR. LANCASTER: I believe Dr. Levy confirmed the foundation as we went over them, sir.

THE COURT: All right. I'll overrule the objections. I'll let in 371, 377, 378, 384 and 374.

(Defendant's Exhibits Numbers 371, 374, 377, 378 and 384 were received into evidence.)

MR. LANCASTER: Thank you, Your Honor. And I have no further questions.

MR. GOODSTEIN: Thank you, Your Honor. Just some brief --

THE COURT: Redirect.

MR. BERNSTEIN: Brief redirect, Your Honor.

THE COURT: All right. You may proceed.

REDIRECT EXAMINATION

BY MR. GOODSTEIN:

Q. Dr. Levy, you were asked about projections for future ambient $PM_{2.5}$ concentrations in counties in North Carolina.

A. Yes.
Q. The current projections for air quality in the future, would you expect those projections to be based on implementation of the Clean Air Interstate Rule?
A. I would expect that that would be the case given that when the modeling was conducted, it was presumed that CAIR would be promulgated.
Q. So with the vacating of the Clean Air Interstate Rule, those projections are going to have to be revisited for the parts of the country that were forecasting improvements resulting from that rule.
A. That's correct.
MR. LANCASTER: Objection, Your Honor. This gentleman's expertise is in environmental risk assessment and public health, not in projecting emissions or -- that's it.
MR. GOODSTEIN: I'm just following up on the line of questioning of counsel to this witness, Your Honor.
THE COURT: Overruled.
BY MR. GOODSTEIN:
Q. And Dr. Levy, do you use toxicological as well as epdemiological literature in your work?
A. I do, yes. I think part of being a risk assessor involves evaluating all different streams of evidence as well as relying on other expert panels and folks who do have toxicology degrees to sift through that evidence and determine

the plausibility of that evidence.
Q. And does the testimony of Dr. Peden, his medical testimony in this case and his reports, support your conclusions regarding biological plausibility of the health endpoints that you used?
A. Yes, it does.
Q. And can you please tell us about the recent ozone study that was issued by the National Academy and what it indicated regarding current mortality estimates associated with ozone.
A. It indicated -- it was a lengthy report, but I think one of the core conclusions from my perspective is it indicated that impacts from ozone would be anticipated including at levels below the National Ambient Air Quality Standards and that therefore the concentration response functions that had been developed by myself and others would be applicable for health impact assessments.
MR. GOODSTEIN: Thank you, Your Honor. We have no further questions of Dr. Levy.
MR. LANCASTER: No recross.
THE COURT: All right. That will conclude your testimony, then, Dr. Levy. You may be excused.
(Witness stepped down.)
THE COURT: All right. Call your next witness.
MR. GOODSTEIN: Thank you, Your Honor. North Carolina calls Dr. Leland Deck.

LELAND BENSON DECK,

being first duly sworn, was examined and testified as follows:

MR. GOODSTEIN: Your Honor, if I may approach, we have a set of exhibits in a binder for Dr. Deck and for the court.

THE COURT: Yes.

(The document was tendered to the court.)

DIRECT EXAMINATION

BY MR. GOODSTEIN:

Q. Good afternoon, Dr. Deck. Can you state your full name for the record, please.

A. Leland Benson Deck.

Q. And what is your current position, Dr. Deck?

A. I am a managing economist with Stratus Consulting, Incorporated.

Q. And I want to refer you to Plaintiff's Exhibit 434 for identification. And is that a copy of your CV?

A. Yes, that is a copy of my resume as I included in my expert witness report circa 2006.

Q. What is your current responsibilities and duties as an economist -- managing economist at Stratus Consulting?

A. I manage and conduct economic analyses, environmental risk assessment analyses for clients that Stratus Consulting has. The majority of the work I have been doing is -- has been on behalf of EPA, on behalf of various state and local

and regional air quality planning organizations, foreign governments also on air quality planning work.

Q. What is your area of expertise?

A. I -- my expertise is in economic benefit cost analysis, specifically in the application of benefit analysis for air quality policy analysis purposes. That includes the estimation, quantification and valuation of health effects, nonhealth effects, et cetera, that will come from improving air quality, mainly ozone particulate matter, visibility related issues to that.

Q. And how many years have you been involved in that work?

A. I entered the field in 1981 when I joined the State of Maryland Department of Natural Resources Power Plant Siting Program. Subsequent to that I completed my Ph.D in Environmental Economics and joined USEPA in 1987. And have been consistently employed explicitly in air pollution benefit evaluation issues since then.

Q. And what has your role been in this case?

A. I was approached by the state to estimate the economic valuation of the human health effects associated with reductions of the excess emissions from the TVA power plants and to compare the economic valuation benefits with the economic costs that the state had also prepared for that.

Q. And can you summarize your educational background which is listed on Page 1 of your CV, Plaintiff's Exhibit 431, for

us.
A. Yes. I have a Bachelor of Science in Geology from Rensselaer Polytechnic Institute, and both a Master's and a Ph.D in Economics from the University of Maryland.
Q. And can you summarize your work experience for us, your professional experience, which is listed in your CV on Pages 1 through 4.
A. Yes. Starting with the State of Maryland, I was working with the state on their periodic report on the environmental and economic impacts of siting new fossil fuel-fired power plants within the state of Maryland.

Beginning with the USEPA, I was working on a series of individual regulations, doing economic analysis of those regulations. While with EPA, those included the setting of the SO_2 ambient standards in 1988, 1989. Those were actually promulgated. Some individual emission source rules and research on methods of doing economic valuation of air quality impacts. Those included visibility valuation impacts and valuing mortality.

When I left OAQPS as my employer and went into the consulting world, my primary client at that point was still EPA, as well as some other state and local and NGO organizations. And while there, I did the quantitative economic valuation, in some cases the risk assessment, of a wide variety of air pollution regulations, including the 1997

PM NAAQS and the ozone NAAQS at approximately the same time. Did the economic benefit assessment and the risk assessment for two major EPA reports to Congress, known as Section 812 reports, referring to a particular section of the 812 requiring those reports. Those are comprehensive benefit cost analyses of the Clean Air Act. The initial report looked at what the country got out of the first 20 years of the Clean Air Act, 1970-1990. The second report looked at what is the country currently and soon to get out of the Clean Air Act, all of its sections, based on the on-the-books and on-the-way kind of actions.

I am currently still working on the next round of the 812 analysis which is continuing that line of comprehensive benefit cost analysis.

During that period of time as a consultant for EPA, I did economic risk and benefit analysis for a variety of major regulations, including the NOx SIP Call, the CAIR rule, the Section 126 Interstate Power Plant rule, the NAAQS themselves, various mobile source emission rules, autos, trucks, a variety of things.

I also worked for as clients various other organizations. I had done for EPA the risk assessment and economic valuation for a proposed piece of legislation that predated CAIR. The administration called it the Clear Skies Act. On behalf of the EPA, I did the legislative analysis similar to a

regulatory analysis, but it was for proposed legislation of that. I was also contacted and asked to do a very similar analysis using the same methods of various alternative bills that were introduced on Capitol Hill at that time so we could do an apples to apples comparison, not only the administration proposal, but one of Senator Carper's proposals, Senator Moynihan's proposal. There was a series of Senate bills that was Senate proposed bills that people wanted apples to apples analysis of those.

Q. So it's fair to say you've done quite a bit of economic analysis of air pollution control programs?

A. That is what I specialize in, yes.

Q. And when you got your master's and Ph.D in economics, that included focus on environmental economics and valuation of environmental effects.

A. Very much. My Ph.D in particular is on one of the important methods used in valuing statistical life. It was a statistical efficiency of the Hedonic Method, and very much working on those valuation issues. In fact, my dissertation adviser serves on many of the peer review boards that I face. I am very tired of the same professor scoring my work. She's been doing this for 30 years, but it's the field we were studying at Maryland and that I continue to work in.

Q. And so you continued to do that work as a senior economist with USEPA?

A. With EPA and then with Abt Associates and now with Stratus Consulting.

Q. Can you tell us about your development, your role in the development of the BenMAP program for EPA. Tell us what that is and what your role was in the development of that.

A. BenMAP is an acronym. It stands for the air quality Benefits Mapping and Analysis Program.

I was a principal investigator in developing that model. That was a -- there was a predecessor model known as the Criteria Air Pollution Modeling System. CAPMS or BenMAP have been used by EPA as the primary tool for their health and economic benefit analysis of air quality rules since approximately 1995. The only difference between the two models, the algorithm, the basic model which I was the lead developer of in CAPMS is the very same algorithm and model used in BenMAP.

EPA decided after the NOx SIP Call that they wanted to make this model available throughout the world, and they asked us to make it a stable -- stable model. It would be run on any Windows application anywhere in the world. Make it usable for the United States, but also anywhere in the world by being able to incorporate population data, air quality data, all of the different moving parts you need to do an air quality analysis.

EPA now has that model as a publicly downloadable model.

They support its use, encourage its use. They offer seminars periodically on how to use it. EPA has sent teams out around the world to train various individuals on -- you know, anyone that wants.

It is a very powerful model. It is designed to be as user friendly as possible. That, perhaps, is in the eye of the beholder exactly how user friendly it is. It's got a lot of different capabilities on handling air quality and population and such to use all of the various permutations and aspects of it. In total it is not thought of as wildly user friendly, but each piece along the way isn't that bad.

Q. Is the approach incorporated in BenMAP for estimating benefits associated with pollution control programs, is that similar to the approach that North Carolina has used in this case?

A. Yes, it is. The overall paradigm that Dr. Levy described is of applying health risk assessments and exposures, and population baseline incidents is precisely what BenMAP does. There are many different computer frameworks that can do exactly the same basic kind of thing. The work that Dr. Levy described he did is exactly the approach BenMAP takes in combining the various sources.

BenMAP makes it easy for a user by having already in it a detailed population base of the U.S. and population forecast and incidents rates and a long library of concentration

response functions that a user can pick and choose as they wish. But it's very much the same explicit method. In fact, many, many of the same materials, the Woods and Poole population forecast. I mean, it's very similar.

Q. Did SAMI use BenMAP for its health analysis?

A. I was involved with the SAMI process and -- which was a multi-stakeholder process. The USEPA asked me as part of their activity in SAMI to take the SAMI air quality modeling analyses that had been done and use the then current version of BenMAP. May have been CAPMS. It's right at that particular transition in time. And the EPA -- EPA's choice of concentration response functions, which I believe was ones used for the NOx SIP Call which had just preceded that. Exact details of which functions are always changing from time to time. And I did the analysis of the main SAMI scenarios that had been -- had undergone, if you will, the full-blown analysis going through the air quality. And so EPA submitted into the SAMI process and is very much part of the SAMI record the EPA methods estimation of the health effects and the economic valuation of those health effects.

BenMAP and CAPMS do the two sides. They both quantify health effects and apply economic valuation to those quantified health effects.

Q. And does BenMAP, the way it works, seek the central tendency?

A. BenMAP can be used in many ways, but yes, that is a primary purpose of it. You can use a specific concentration response function, either from the library that is included or you may add your own, which is a big advantage when a new piece of research comes out, what we sometimes call the study of the month comes out, you can immediately put that new result in and use those results. It does produce the central tendency estimates or using the mean value of those distributions, of that parameter, the risk coefficient parameter. It can also do a wide variety of uncertainty analyses, meta-analytic type analyses in a number of ways.

As I said, it's a powerful program with a lot of different uses. In all cases, the central tendency or mean value is a very featured output of it that's very -- the output is easy to find because in many cases that's what the user is interested in.

Q. And BenMAP has been published and peer reviewed by the National Academy?

A. It has been throughout its development step by step. It was peer reviewed by EPA Science Advisory Board. So the methods that go into it in any number of different aspects, concentration response, exposure, handling of the air quality data, population forecast, each of those steps was reviewed by EPA Science Advisory Board over the course of time, so it was a periodic review.

The -- in 2002, if I remember right, the NRC published a book Reviewing EPA's Benefit Cost Analysis. And in that they did four case studies of particular regulations, the PMX and NOx SIP call, one of the mobile source rules -- the fourth one escapes me at the moment, where BenMAP was used as the tool. It was my analysis that EPA used in that so at that point it was reviewed by the NRC.

The NRC also just completed, Dr. Levy alluded to it earlier, about the -- in their review of modeling estimating ozone risks, they included a review of EPA's methods for applied risk assessment for ozone, which again, is BenMAP -- is using BenMAP as EPA used in the analysis in the national risk assessment and the regulatory impact analysis that they used for the 2008 ozone NAAQS resetting.

Q. Have you done economic analysis of air pollution controls on coal-fired power plants previously?

A. Yes, indeed. Much of my work throughout has included power plants. Many of the individual papers and studies in my CV are -- some of them are explicitly coal studies or coal-fired power plant studies. Others, such as the NAAQS, are studies of regional control plans which control power plants and others. So there's a range of single plant analyses, collections of plants. The SAMI study, kind of a region wide study of power plants. My study for the Great Lakes Air Directors Consortium, which is a regional planning

organization of five states surrounding the Great Lakes, was of their power plant control options.

There are -- actually for the EPA did a -- involved when I was an employee there of a major study of one single power plant in Arizona that's owned and operated by the Salt Lake -- by the Salt River Valley Project that owned a generating station that became a well-known power plant control -- control study. I was fortunate to receive a Gold Medal from EPA for my role in that benefit cost analysis of that power plant regulation. Lots of power plants.

Q. All right. And your publications are listed at Pages 4 to 8 of your CV?

A. Yes, they do. And those include articles in books, chapters in books, published articles on both methods and applications of the methods of doing this. And a number of reports done for the government as a contractor or where the government has incorporated in part or wholesale my analyses in their -- in their technical support documents for any of a number of rules, many of which I've mentioned here today. There is also, again, reports for other states, locals, NGO's as well.

As this resume is moderately dated in 2006 and I continue to earn a living doing this, there are more projects that I have going on. A major one I did since the time of this was for the South Coast Air Quality Management District, which is

the greater Los Angeles metropolitan area. I did the health assessment and economic valuation of their future control plans for ozone and PM. Very similar styled analysis to what we're talking about here. They ran their quality modeling and they did that analysis. That analysis is closely tied to the CARB report that Dr. Levy mentioned earlier today. CARB is an obviously connected regulatory agency to the South Coast. They were very involved in the South Coast methods development as South Coast was in the CARB plan development. It's not real surprising that South Coast's report last year and CARB's report this year use essentially the same selections of valuation techniques, of concentration response functions as I used in the South Coast.

Q. And you're a member of some professional associations in the field of economic analysis and you're also peer reviewer on a number of publications?

A. Yes, I am. I'm a member of the American Economics Association, Association of Environmental Resource Economics and Waste Management Association, the Society for Risk Assessment.

I routinely do peer reviews for all of the journals out of those four organizations. Several of them have multiple journals. I continue to do peer review for the Review of Economics and Statistics where my dissertation got several publications in. And I was also a peer reviewer for a

National Research Council recent book on risk assessment methods. I was a peer reviewer for that.

MR. GOODSTEIN: Your Honor, we tender Dr. Deck as an expert in economic analysis of air pollution controls.

MR. LANCASTER: And we've reached a stipulation on that, Your Honor.

THE COURT: All right. Let the record show that the court so holds.

BY MR. GOODSTEIN:

Q. Dr. Deck, have you prepared some expert disclosure reports in this case?

A. Yes, I have. And there have been three reports: My original expert report, a supplemental expert report and a letter report.

Q. And are they identified at the back of your notebook as Plaintiff's Exhibit 477, 478 and 479?

A. Yes, they are.

MR. GOODSTEIN: Your Honor, at this time we offer 477, 478 and 479 into evidence.

MR. LANCASTER: Our only objection are to the portions which value health impacts in the 33 state areas instead of North Carolina.

THE COURT: All right. Show the objection overruled. And the court, as has previously been indicated, will review the -- include the surrounding areas, but be

concerned about the states named.

MR. LANCASTER: Thank you, Your Honor.

Q. Dr. Deck, were you able to estimate the value of the health benefits associated with the emissions reductions on TVA power plants sought by North Carolina in this case?

A. Yes, I was.

Q. And what was your overall conclusion?

A. Using the -- valuing the estimated health effects that Dr. Levy testified to few minutes ago and using the standard current peer-reviewed valuation techniques, I estimated that the aggregate benefits for using the 2000 population are about $9-1/2 billion total; and using the 2013 population, nothing else changes, same air quality, just updating the population which is larger as Dr. Levy did, comes to $10.9 billion. That is for the single year 2013 air quality emission reductions.

Q. And it's your conclusion that you would expect those types of benefits to accrue for each year that the emissions are reduced from TVA power plants as requested by North Carolina in this case?

A. Yes, I would. And in fact, going forward in time beyond 2013 as the population throughout the region increases, the health effects would increase along with the population and hence the economic valuation of those health effects would also increase year by year merely tracking along with the increasing population.

Q. Now, are these values comparable to the economic impact of TVA's emissions today?
A. Yes, they are. As we previously heard testimony earlier in the week, that the excess emissions from TVA's plants are -- in 2013 are comparable to the 20 -- to the current excess emissions in, say, 2006 and the health effects would be comparable then to those excess emissions; and finally, the valuations would be comparable to 2006, the aggregate value in 2006 would be comparable to what I estimated in 2013.
Q. And did you also compare these benefits that you've estimated to the cost of the emissions controls and the emissions reductions sought by North Carolina in this case on a yearly basis?
A. Yes, I did. I used the -- I took the cost estimates, actually three different cost estimates that the state provided me from Dr. Staudt. And because those cost estimates included both a capital component, the upfront investment cost to install all the equipment, and the operating and maintenance costs, the annual flow, I had, using standard economic techniques, to convert those into an annualized basis. So it's effectively one year total costs of operating the plants -- the control equipment, so I could compare that with the one year benefits to put it on an apples to apples comparison.
Q. And what was your overall conclusion regarding the

benefit cost ratio associated with the emissions reductions from TVA power plants sought by North Carolina in this case?

A. Using a single cost estimate just for simplicity here and not get lost in numbers, where the $4.2 billion cost -- investment cost, that comes out to $638 million annualized costs. Comparing that to the benefits throughout the region of $9-1/2 billion versus $638 million costs, it's approximately a 15 to 1 ratio. Benefits were 15 times the overall -- 15 times the costs.

Looking at just the benefits in North Carolina, using the health effects from just North Carolina that Dr. Levy presented, that benefit cost ratio comes out to be 1.05. So the benefits in North Carolina alone covered the costs.

And there's certainly information for each -- each state and you can run all the permutations about, you know, what the benefit cost ratios would be, including any combination of states that you're interested in. I do present benefits by state throughout allowing you to do it any way you want to.

Q. All right. And what method did you use to calculate the monetary value of the benefits to North Carolina in the region of the emission reductions for TVA plants sought by North Carolina?

A. The overall approach is called a damage function approach. We've been using this -- Dr. Levy was using this also. You look at the benefits piece by piece for each health

endpoint. And in concept you would go on and then look at each category of nonhealth effects. And so piece by piece and then you add up the sums. That's called a damage function approach.

So for each of the health endpoints that Dr. Levy estimated, I used a valuation technique that comes down -- that's really what's called a unit value. A value for each of those hospital admissions, for each of those premature mortalities, school absence, and just for each and every one of those health effects there is a method and -- that comes up with a unit value for each of those health effects.

Then at the end it becomes sort of simple multiplication of a value for health effect times the number of health effects is the aggregate value of that health effect and then you sum across the health effects.

Q. Did you prepare a summary of the unit values that you used for each of the health effects that you monetized?

A. Yes, I did.

Q. And can I refer you, please, to Plaintiff's Exhibit 383 for identification.

A. Yes, that is --

Q. Can you identify that for us, please.

A. Yes. This is a table that this information appears in a table in my expert report. These are the unit values for each of the health effects that Dr. Levy testified to previously.

Q. And can you describe what this summary shows.
A. Yes, indeed. There's a lot going on here. There's a separate story, if you will, separate method for each of the -- each of the line items. Clearly the mortality one is perhaps the most important. It dominates the aggregate benefits.

What an economist -- so these are showing the initial point is a 5-1/2 -- $5.5 million number used for adults and infants. I'll probably be talking about this value a good bit and I want to point out that the $5-1/2 million is measured in 1990 incomes and 1999 prices. It can get confusing as we adjust for -- as income changes and the inflation goes on, but for -- as we see here, going from the column with 5-1/2 million, just adjusting it for the inflation that's going on between then and 2006 as well as real income growth, not just prices going up, but real income growth, that $5-1/2 million base price, if you will, base value, goes to $7.25 million.

The income -- the price adjustment is very straightforward. It's using the consumer price index such as social security uses to adjust -- I mean, it's a very commonly used inflation.

As real income goes up, though, as human beings our demand for things we want goes up. As we become wealthier as a nation in real terms, we want more healthcare. We want more education. We're willing to pay more for protection against

risks. So there is an established method where as income goes up, values or demand goes up. Not on a one-to-one basis. Income goes up 1 percent; demand goes up at a slower rate, but it does go up. Combination of those two get us to $7.25 million as a value of statistical life. That is what's used for both infant and adult premature mortality.

There is a wrinkle on the adult premature mortality. The air pollution from a single year is modeled to cause premature mortality. EPA has developed an explicit assumption that although the one year's emission reductions cause premature mortalities, they don't all occur in exactly that year. The majority of them, say half of them occur within a one year period, but then the rest kind of stretch out over a period of time. Most in one year, I think it's 3/4 or 80 percent total within the first five years and then a long tail still associated with that one year emission reduction.

To an economist, events still coming on at different points in time, we are an impatient species. We would rather have things now than later, so those mortalities that are manifesting themselves 20 years after are worth -- our willingness to pay for them is slightly less than it is up front. Therefore, that is called the lag structure of adult premature mortality stretching out over 20 years ends up with a net effect of the unit values for adult premature mortality of 6.5, 6.6 million dollars only because of that PM lag

effect.
Q. All right. And you used the EPA unit values for these various health inputs?
A. Yes, indeed. What an economist would like to value in general -- this is not a value of a life as is often used in certain courtroom situations. In a common courtroom situation you're talking about after someone has died, talking about the damages, perhaps an economic value for that particular individual. That's an after-the-fact valuation.

We are talking here about before-the-fact risk reductions, ex-ante reductions. There is a large body of economic research that has gone on in that. There are literally hundreds of studies of what the value of the risk reduction is. That's the real underlying question here. What are people willing to pay to reduce the risk of dying.

The $5-1/2 million really comes out of the consensus of all of the literature is that for a small change in risk of mortality, for a small one in -- let's say a one in a million reduction in the risk, economic research has found the central estimate is people will pay about $5-1/2 for that. There is -- and if a million people are exposed to that -- or enjoy that opportunity, a million people with a one in a million risk, that's one statistical life, and that million people would have each offered to pay or would have paid $5-1/2 and now it comes up to $5-1/2 million for the one statistical

life.

EPA actually now follows OMB guidance on what the value of statistical life to use is. That guidance is a range of 1 million to 10 million dollars. That's OMB's 2003 guidance. $5-1/2 million is the central -- it's the midpoint of that 1 to 10 million dollar range.

There's a lot of peer reviews going on since then, but, yes, this is OMB's guidance as EPA applies it and the rest is just a unit -- you know, the 2006 prices in incomes adjustments off of that -- off that current government guidance.

Q. And you discussed the lag structure associated with adult premature mortality.

THE COURT: I think we'll stop now for lunch, Dr. Deck. We'll come back and take up at that point. 2:30.

(Lunch recess at 1:17 p.m.)

UNITED STATES DISTRICT COURT

WESTERN DISTRICT OF NORTH CAROLINA

CERTIFICATE OF REPORTER

I certify that the foregoing transcript is a true and correct transcript from the record of proceedings in the above-entitled matter.

Dated this 18th day of July, 2008.

s/Cheryl A. Nuccio
Cheryl A. Nuccio, RMR-CRR
Official Court Reporter